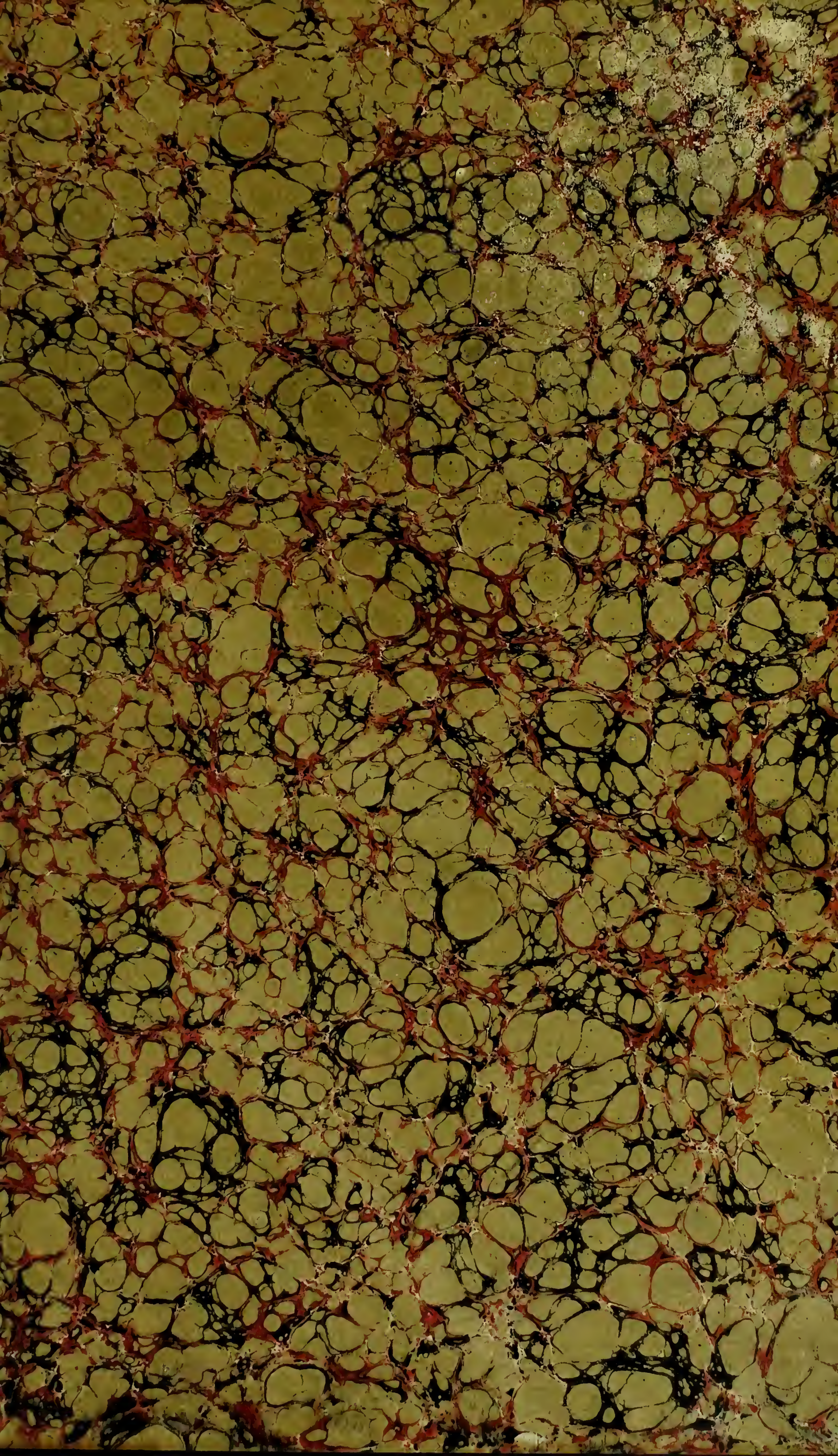


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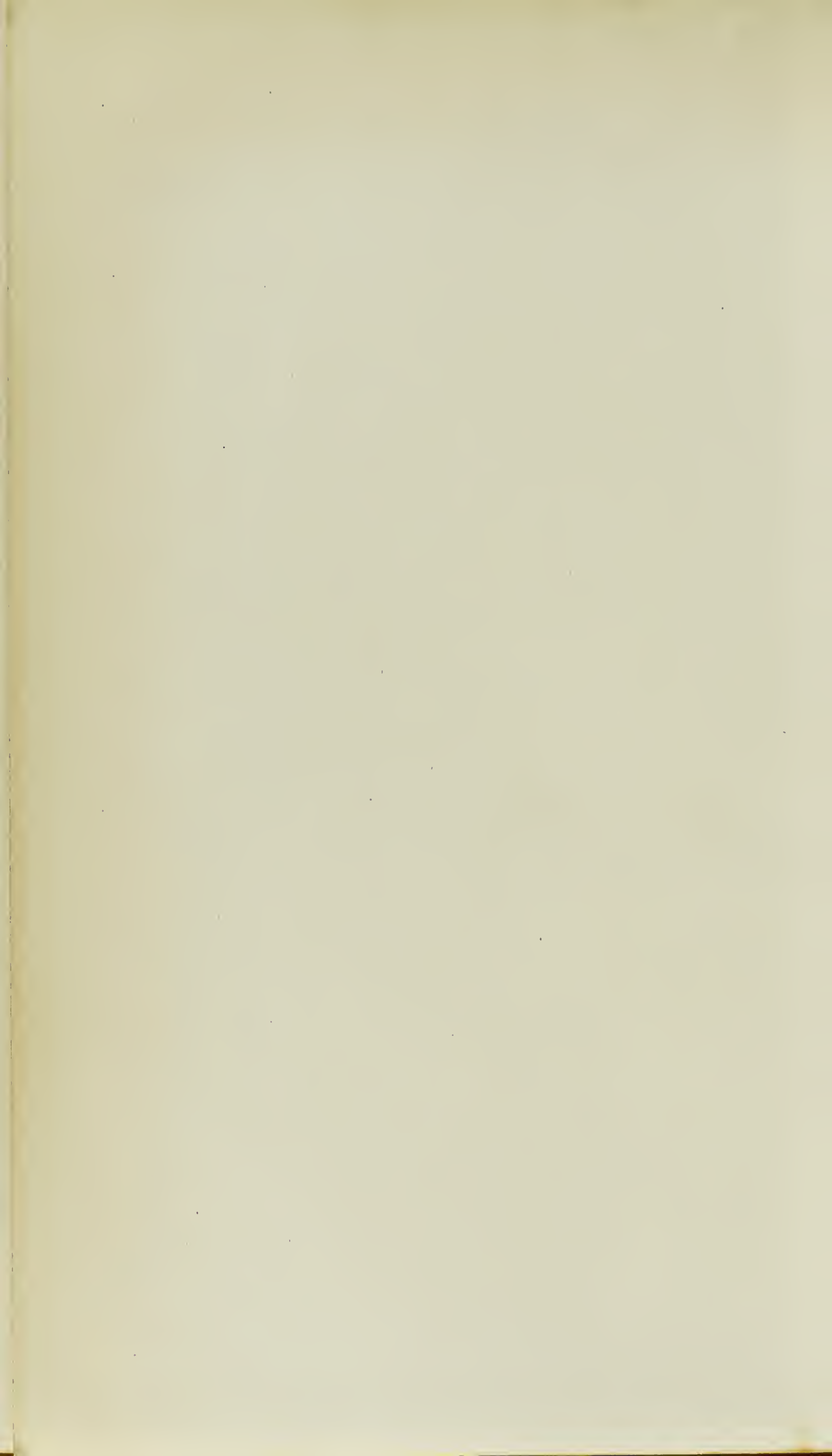




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Presented by Thomas Watson, M.D.
YELLOW FEVER
President of the College.
1854.

CONSIDERED IN ITS

HISTORICAL, PATHOLOGICAL, ETIOLOGICAL,

AND

THERAPEUTICAL RELATIONS.

INCLUDING

A SKETCH OF THE DISEASE AS IT HAS OCCURRED IN
PHILADELPHIA FROM 1699 TO 1854.

WITH

AN EXAMINATION OF THE CONNECTIONS BETWEEN IT AND THE FEVERS
KNOWN UNDER THE SAME NAME IN OTHER PARTS OF
TEMPERATE, AS WELL AS IN TROPICAL, REGIONS.

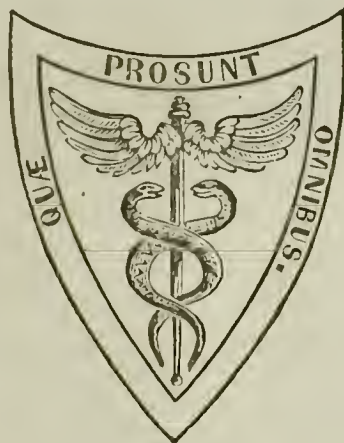
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TURIN, COPENHAGEN, STOCKHOLM, NANCY, AND NEW ORLEANS; OF
THE MEDICAL SOCIETIES OF NAPLES, MARSEILLES, LYONS, ETC.

IN TWO VOLUMES.

VOL. I.



PHILADELPHIA:
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1855.

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TO
HUGH L. HODGE, M. D., AND JOHN BELL, M. D.,
OF PHILADELPHIA;

A. P. MERRILL, M. D.,
OF MEMPHIS, TENN.;

S. D. GROSS, M. D.,
OF LOUISVILLE, KY.;

AND

JOHN TRENOR, M. D.,
OF NEW YORK;

These Volumes are Inscribed,

AS A

TESTIMONIAL OF THE HIGH RESPECT AND REGARD

TO WHICH

THEY ARE SO JUSTLY ENTITLED FOR THEIR PROFESSIONAL EMINENCE,

AND AS A

TOKEN OF THE SINCERE AFFECTION ENTERTAINED FOR THEM

BY THEIR FRIEND,

THE AUTHOR.

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P R E F A C E.

THE subject of yellow fever has long attracted the attention of the medical profession on this and the other side of the Atlantic. Upon the origin, the mode of propagation, the pathology, and the appropriate treatment of that fatal disease, as much, perhaps, has been written, particularly within the last sixty years, as upon any other malady flesh is heir to. The medical literature of England and France is rich in works of high value on these topics, both as regards didactic treatises on the disease considered in its totality, or monographs on its several branches, and descriptions of separate epidemics observed in Europe and tropical regions; while Spain, Italy, and, to a more limited extent, Germany, have contributed valuably to our stock of publications on the same subject.

Of all countries situated beyond the limits of the tropics, none has been so frequently visited by, or has suffered so severely from, the disease as our own, in some portions of which it may be viewed as assuming, in great measure, the character of a true endemic. But while such is the case in New Orleans, Charleston, and other southern points of our extensive coast; while we almost annually find that unacclimatized strangers are there attacked at the usual season; and while the disease, after short intervals of repose, assumes the form of a more or less diffused and fatal epidemic, many of the Atlantic cities and towns of our middle States, and a few of the more northern ones, though seldom the seat of even sporadic cases, have been at times more or less scourged by epidemical manifestations of the disease.

Among the places thus visited, the city of Philadelphia has, doubtless, to an intense degree, suffered from the calamity in question. Soon after its settlement, it was severely afflicted by the fever under consideration; and from that epoch to the present time, embracing a period of little more than a century and a half, it has been the seat of many such visitations, the result of which was, on some occasions, of the most destructive character. The accounts handed down to us of the extensive diffusion of the disease during some of those epidemics, and of the mortality accruing therefrom, afford, indeed, pictures as appalling as anything recorded respecting other American cities, and but little, if at all, inferior to the results of the memorable pestilence of Andalusia in 1800, and of Barcelona some twenty years after.

As may be presumed, the medical writers of this country, alive as they are to every subject of interest and importance in a professional point of view,

and especially to one which—for various reasons, I shall advert to as we proceed—involves questions of much public interest, have not remained silent in reference to the disease which forms the subject of the present inquiry. From the days of Lining and Moultrie to our own, numerous writings, of various degrees of pretension in point of size and merit, have accumulated on our shelves; while the medical periodicals of the country contain a goodly number of valuable communications on the subject.

Among those who, on this side of the Atlantic, have contributed most largely and valuably to the stock of our knowledge on this important disease, the physicians of Philadelphia, from an early period of the professional history of this city, have occupied, to say the least, an eminent rank, both as regards the number and the merit of their publications. Each of the epidemics by which the city has since been visited, and including the fatal one of 1793, has had one or more historians; whilst detached volumes or essays have appeared on the pathology, the origin, the mode of propagation, the treatment, and the prophylaxis of the disease.

In view of this fact—in view of the number of valuable works we possess on these topics—the author of the present volumes would surely have abstained from adding another, much larger than any of its predecessors, to those already in the hands of the profession, had he not thought that a work embodying a historical summary of all the epidemics of that fever which have occurred in his native city at various times, from 1699 to that of last year, as well as a *resumé* of our knowledge of the nature and causes—known or presumed—of the disease, such as it has appeared on those different occasions, might prove acceptable to medical readers curious in such matters, if not to the profession at large. A work of this particular kind has not yet been undertaken—so far, at least, as the author has been able to learn; but it cannot fail to prove useful; for the professional reader who limits his researches, as is but too often done, to the descriptions of the more recent epidemics, or to a few monographs on the disease generally, loses the recollection or fails to become acquainted with the events of former days, and thereby misses the opportunity of comparing together the circumstances under which the several visitations have occurred, as well as the character the disease presented and the treatment it required, and of reaping all the advantages naturally accruing from examinations of the sort.

It is to supply this deficiency that the present work is offered. It was originally prepared in a foreign land and in a foreign language, for a special object which was never attained, and is now issued at the urgent request of several professional gentlemen, whose friendly regard for the author has led them to form a higher estimate of his capacity to perform the task than he probably deserves. Be this, however, as it may, the work is presented, after undergoing such alterations, additions, and amendments as a greater familiarity with the subject and a more careful examination of the materials obtained demanded, to say nothing of the greater facility of acquiring knowledge on the various topics passed in review which a constant access to a very large collection of publications and documents on the yellow fever—from the tiny

pamphlet to the fat and stately quarto, or even folio—has enabled the author to enjoy.

Fully aware of the justness of the remark of a great master, that

. “quiconque ne voit guère
n’a guère à dire aussi,”

the author has neglected no opportunity of rendering himself practically familiar with the disease of which he had undertaken to become, as it were, the historian. With a vivid recollection of the many cases he had occasion to observe in a former epidemical visitation, and subsequently, when the disease showed itself in a sporadic form, he not unfrequently enjoyed the opportunity of noticing it elsewhere—even beyond the limits of this country—though more particularly in this city during the summer and autumn of 1853 and the year following. On all these occasions, nothing connected with the causes, nature, and treatment of the fever has been allowed to escape a careful scrutiny. The result has been that, though a smaller number of cases may have passed through the hands or under the eyes of the author than falls sometimes to the lot of other physicians, in places frequently visited by the disease, he has seen enough of it, in its diversified aspects, and investigated what he has witnessed with sufficient minuteness and care, to justify him in presenting, in the present form, the result of his personal observations, and comparing them with those of other writers.

The occasion seems most opportune, inasmuch as the revival of the disease among us a short time ago, after a repose of many years—its diffusion over a large expanse of this country; its very general occurrence, at the same time, in tropical regions; its appearance, during the last few years, in localities where it had never existed, and in spots where it was thought it could never reach; as also the frightful loss of life it has almost everywhere occasioned—have once more imparted to everything connected with the subject a degree of interest which it had in some measure lost.

But the work, though treating more especially of the yellow fever of Philadelphia, aims at something of a more enlarged and important character. By means of a constant comparison of facts observed here with those noticed elsewhere, on every point connected with the disease, the author has been enabled to lay before the reader an *exposé* of the whole subject; in a word, a comprehensive treatise on the yellow fever, in which every point of interest is fully examined. The fever of Philadelphia is used, in what follows, as a kind of foundation to a full consideration of the disease, as observed in different places, at home and abroad, where it has occurred—as a central point, towards which facts connected with the fever, as manifested elsewhere, converge; in other words, to use a homely expression, as a hook upon which to hang a dissertation on yellow fever generally—its symptomatology, its anatomical characters, its pathology, its treatment, its etiology, and the laws by which it is governed.

Those of the readers of the present work who have had the patience to peruse a volume the author laid before the public some eighteen months ago,

will doubtless recognize, as they proceed, many a passage and statement they had seen before. It is proper he should remark that the few pages of that volume which have found their way into the publication now offered, have not, strictly speaking, been borrowed from the other. They are, in truth, placed where they properly belong, having for the most part been extracted from the manuscript of the present work, as being suitable to illustrate and sustain the etiological and pathological views the author was then endeavouring to enforce.

In undertaking to produce a medical account of the yellow fever, as it has appeared in Philadelphia, as well as an historical sketch of the various epidemics by which that city has been visited, from the time of its settlement to the present day, the author has thought, and others, in whose judgment he confides, have entertained the same opinion, that it would be advisable, for the better illustration of the different topics embraced in the inquiry, and upon which it will be necessary to enter somewhat in detail—the probable or ascertained causes of the disease; its mode of progression, from the outset to the close of each visitation; the nature of the localities invaded; the thermometrical and hygrometrical conditions under the influence of which the disease appeared; as well as its mode of propagation, and the extent and proportionate amount of mortality it occasioned—to lay before the reader a brief description of the extent and physical condition of the city at the several epidemical periods, including its topography, climate, population, hygienic peculiarities, &c.

Deprived of the facilities afforded by such an introduction to the main object in view, it would be difficult, if not impossible, for the reader, considering that the history of the disease covers a period of more than a century and a half, during which Philadelphia has expanded from a small town to the broad dimensions of a city of undoubtedly second rate order, not only to follow the narrative given of the rise and progress of the fever, but to form a comprehensive idea of the various circumstances adverted to. He would be unable to connect the advent of the disease with the nature of the localities visited and the meteorological phenomena characterizing the period; and to compare the fever, considered in its sporadic or epidemic character, as it has been observed here, with the same disease as it usually shows itself in other sections of this country, in Europe, in the West Indies, and on the coasts of South America and Africa. Nor would he ascertain how far the city will compare, in respect to its climatic characters generally, with other places visited occasionally or habitually by the disease.

Unless possessed of the information conveyed by these preliminary details, the readers, especially those who are unmindful of the extraordinary changes which the plot the city stands on has undergone during the period in question, might fancy that the conditions of the place were the same at each epidemic return of the fever. While changes in all these respects have occurred in this city, to an extent unheard of in countries influenced by circumstances different from our own, and where physical transformations proceed with less rapidity than with us, the population, exposed to the

ravages of the disease, has, as a matter of course, experienced modifications of equal magnitude; and conclusions relative to the proportionate mortality, based on the amount of deaths at one time, would find no application to the events of other periods of calamity. Nor is it less evident that, on all these topics, as also in regard to topographical, climatic, and other features; to soil, position, distribution, and mode of construction, &c., the city offers peculiarities, in reference to some of which it has also undergone modifications of greater or less importance.

The author cannot but hope that medical inquirers who aim at taking something beyond a cursory survey of the subject, and wish to reach correct conclusions respecting the characters of the yellow fever generally, the causes to which it may, with the greatest degree of probability, be ascribed, and the various influencing agencies which may contribute to modify its diffusion and its effects, will recognize the propriety and usefulness of the course he proposes to pursue.

In conclusion, he begs to be allowed to express his grateful acknowledgments to his friend, Dr. S. Lewis, for his valuable assistance in the preparation of the Bibliography of the disease, prefixed to the work. Without the aid of one well versed in matters of the kind, it would have been impossible for the author—labouring, as he did, under the pressure of multifarious occupations growing, in part, out of the printing of so extensive a publication—to extend that Bibliography to its present dimensions, and thereby impart to it a desirable degree of completeness. The author must also return his thanks to another friend, Dr. D. P. Lajus, who was kind enough to assist him in framing the tables of mortality contained in the first volume, and to verify some of the calculations therein contained.

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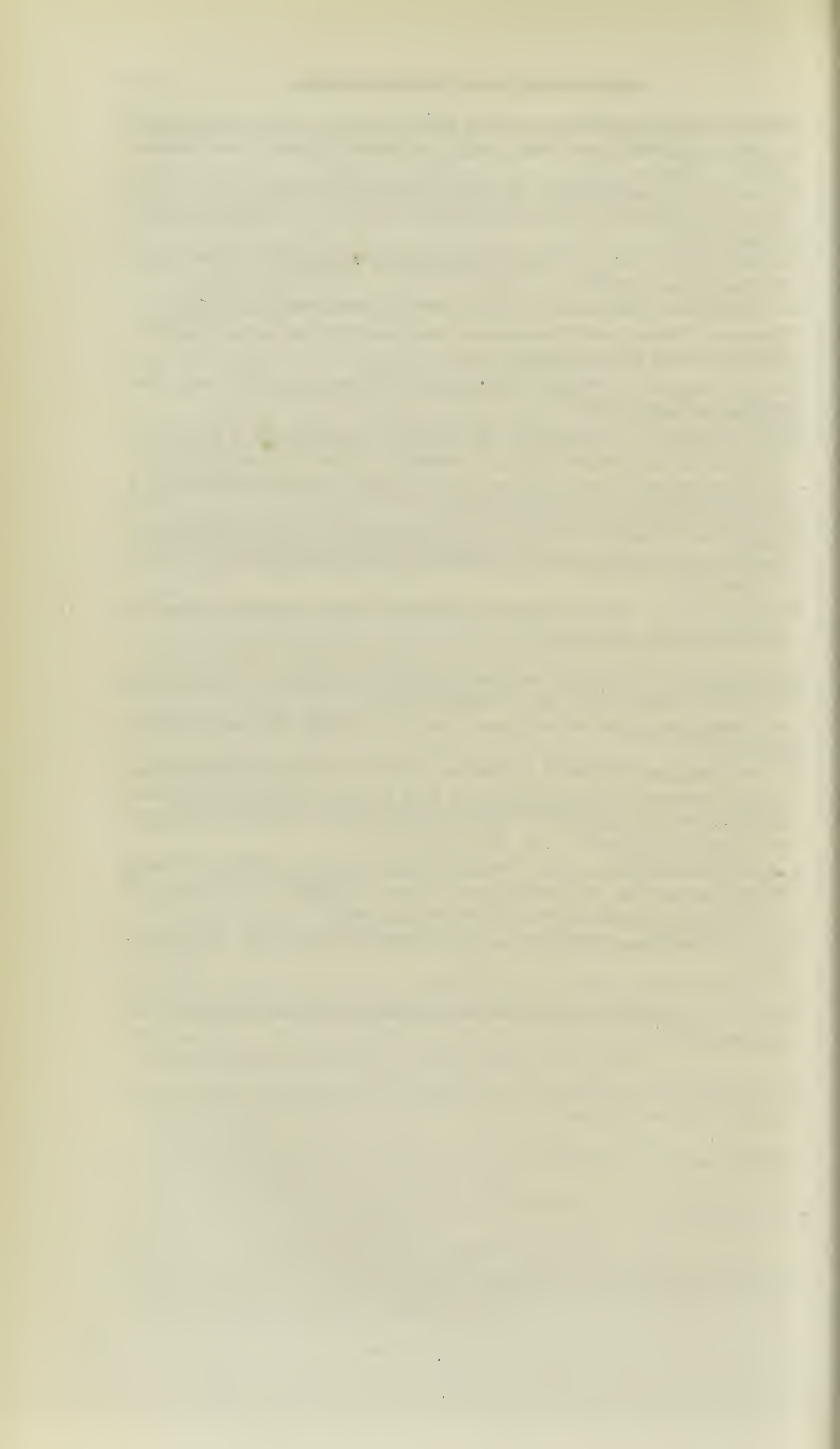
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YELLOW FEVER.

A

T R E A T I S E

ON THE

Y E L L O W F E V E R .

PRELIMINARY OBSERVATIONS.

MEDICAL TOPOGRAPHY.

TOPOGRAPHY.—The city of Philadelphia is situated on a plain, at present nearly level, and on the western bank of the river Delaware, in $39^{\circ} 57'$ of north latitude, and $75^{\circ} 8'$ of longitude west of London.¹ It lies at a distance of about one hundred and twenty miles from, and north of, the Atlantic Ocean by the course of the river; sixty miles in a direct line to the mouth of the latter, and forty-eight miles due west over the State of New Jersey. In accordance with the plan traced by the founder of Pennsylvania, the city extends from the Delaware westerly as far as the river Schuylkill—a distance of about two miles—the latter river being a beautiful stream of water, about two hundred yards wide, and admitting sea vessels of moderate size. It originates at a considerable distance in the mountainous district of the State, and loses itself in the Delaware a few miles to the south of the city. The city proper occupies a space of little more than a mile along the margin of the Delaware. But, such has been the growth of the northern, southern, and western suburbs or districts, with which it has lately been consolidated, that the improvements at present cover a space from north to south of several miles. Towards the centre, as we proceed westerly, the improvements are of a somewhat less compact character; while along the Schuylkill they are still less expanded, leaving very many open spaces between them.

The portion of the isthmus on which the city is located is of modern tertiary formation, consisting of sand and gravel, for the most part overlaid with a thick stratum of clay of various hues and degrees of tenacity—the whole resting upon a primitive basis, which lies at the distance of from forty-five

¹ Others say $39^{\circ} 56' 59''$, long. $75^{\circ} 9' 54''$. Others, again, $39^{\circ} 57'$ lat. north, and long. $1^{\circ} 54'$ east of Washington. “ $39^{\circ} 57' 9''$ lat., $75^{\circ} 10' 37''$ long.” (High School.)

to fifty feet beneath the surface, but shows itself in some of the northwestern districts. "That it was once," says Dr. Emerson, "covered by the sea, is clearly shown by the marine deposits, consisting of bones, teeth, shells, &c., found below the natural surface. Vegetable relics have likewise been met with. A few years since, hickory-nuts were discovered, in digging a well, at a depth of more than thirty feet. In one of the highest points of the city plot, the trunk of a buttonwood (*Platanus occidentalis*) was found imbedded in a black mud, and surrounded by acorns and leaves."¹ Water is easily obtained in every part of the city plot. Near the rivers, it is found at a depth of ten or twelve feet from the surface; in other situations it is found at thirty feet. The subterranean sources which supply the springs under the city have no connection with the Delaware and Schuylkill, as they lie above their high water-mark even in spring-tide. The plateau upon which the city is located is at present flat, and nearly level, with a gradual slope from the centre to each river. The elevation of the soil above low water-mark varies from two to forty-six feet, with an average, according to the late Dr. Rush, of forty feet; the most elevated portion being about the third of the way between the Delaware and the Schuylkill. On the northern side of the city there are several ridges and hills, which, in the progress of improvement, have been much reduced. In a northwestern direction, the land is comparatively high, as also along the margin of the Schuylkill; while, on the western side of that river, it rises to a considerable elevation. The greater portion of the country in those directions is flat, or rather level; little wooded, well cultivated, and spotted with neat and even fine cottages and dwellings. On the southern side of the city, the tract of land comprised between the latter and the point of junction of the Schuylkill with the Delaware, and which has received the name of the Neck, is low, flat, and marshy. The greater part of it is meadow ground, the seat of fine vegetable gardens, from which the city is plentifully supplied. The banks of this small tract are subject to overflow, against which embankments are in some places thrown up.

The Delaware, opposite to Philadelphia, is about one mile in width, and of sufficient depth to admit vessels of heavy burden. It rises and falls with the tide between five and six feet in common times; but, during a long continuance of violent north winds, it occasionally reaches a height of from seven to nine feet beyond its common level. It flows at the rate of from two and a half to four miles an hour; the tide running seven hours up and five down.² The water ceases to be brackish at a distance of thirty miles below the city, opposite to which it is of an excellent quality. The Jersey shore, opposite to the city, is quite flat, dry, and, like the whole of the lower portion of that State, sandy. Between it and the city lies a small island.

The city of Philadelphia, considered in its totality, is undeniably one of the neatest, handsomest, and cleanest in all America. The streets are, for the most part, straight, wide, and well aired; and, with three exceptions—two

¹ Emerson's Stat. of Phil.; American Journal Med. Sci., i. p. 118.

² Mease, Pict. of Phila., p. 16; Rush, Account of Pennsylvania, p. 5.

of which are beyond the limits of the city proper—intersect each other at right angles. These streets are properly graded and paved; they are furnished with wide and commodious side-walks, which, by dint of frequent brushing and scrubbing, are kept in an unusual state of cleanliness. These side-walks are paved with brick—in some places with flagstones—raised on a level with the highest part of the street, and defended from the approach of carriages by ranges of curbstones. Along the external edge of these side-walks, and between them and the paved street, are placed gutters of sufficient width and depth, by means of which the water and filth find an easy access to the river. In some parts of the city, where it has been found impossible to obtain a sufficient declivity, or where, from local circumstances of a peculiar character, the accumulation of water is generally or occasionally very considerable, large circular sewers have been constructed under the pavement of the streets. Into these sewers the gutters of the adjacent streets discharge their contents through apertures placed at convenient distances. These sewers empty into the river, and are washed by rains, though more particularly by the water of the river which flows into them at every high tide.¹

In the very large majority of cases, the houses are built of excellent bricks, from two to four, generally three stories high, with convenient cellars under them, and yards behind. Dwellings of more recent date are sometimes provided with well-constructed water-closets, either in the main or in the out or back building; but much more commonly the privies are situate at the furthest end of the yard. The sink of these privies is dug sufficiently deep to reach the water. It is built round with bricks or stones, and securely covered; and is provided with an air-pipe, which rises a few feet from the surface and passes through the roof.

The city is supplied with an abundance of water from the river Schuylkill, by means of works which have acquired a justly merited reputation for beauty, simplicity, and effectiveness. These works convey the water through iron pipes over the whole city and the larger portion of the suburbs, each house of which is furnished with one or more pumps or hydrants. Besides these private hydrants, there are public ones in the streets, as also plugs, made use of in case of fire, or for the purpose of watering and cleansing the streets. The water is of excellent quality and highly wholesome, containing, according to Professor Boyé, only 4.480, and according to Professor Silliman, 5.500 grains of foreign matter in one gallon; the difference being in the organic matter, which necessarily varies with every freshet in the river. The extent of pipes to convey this water covered, in 1847, a surface of seventy-five miles in the city proper alone; while the quantity distributed to the city, and the districts of Southwark and Moyamensing (the district of Spring

¹ The edges of the pavements are planted, in many streets, with trees. In former days, these trees consisted mostly of Lombardy poplars, for the introduction of which the city was indebted to William Hamilton, who brought them over from England in 1783. They have now been superseded by the lindens, the maple, &c., which afford more shade, are handsomer, and not, as the former, injurious to the pavements.

Garden deriving its supply from a separate establishment), was, on an average throughout the year, 4,075,682 gallons per day; equal to 179 gallons to each tenant, or 30 gallons to each individual.

But, although the general character of the streets is such as described above, there are a few exceptions to the rule, which must be noticed in this place. The first of these consists in a street which plays an important part in the history of most of our yellow fever epidemics. It runs along the margin, and lies under the bank of the Delaware River from one extremity of the city to the other. The situation of this street, owing to the height of the banks, is low, confined, and imperfectly ventilated. It is narrow, and compactly built with high houses, not one of which, in at least two-thirds of the extent of the street, has a single foot of yard room, the privies being placed in the cellars. For many years after the settlement of the city, this street, on both sides, was thickly inhabited by the better classes of people; but for some time past, the houses have been in a great measure converted into stores or shops, while the balance are tenanted by the poorer and lower orders—sailors, emigrants, &c. As may be presumed, from its situation under a high bank, its mode of construction, the use to which it is appropriated, its proximity to the river, the character and number of its occupants, this street is far from being kept in that state of cleanliness so necessary to the preservation of public health; and is withal imperfectly ventilated.

It is but justice to the philanthropic and enlightened founder of Philadelphia to remark, that the street which, as we have seen, constitutes an unfortunate exception to the rest of the city, formed no part of the original plan he had in view. The space it now occupies was left as a convenient road of communication from what was denominated the Penny-pot Landing, at the northern end of the city, to the Blue Anchor, towards its southern extremity; the high bank along the Delaware preventing access to the westward at other places. (*Mease*, p. 23.)¹

The other exceptions to which I have alluded as regards the general arrangement, convenience, and healthful construction of the city, consist in a number of alleys, closed courts, lanes, or narrow streets, by which the blocks of buildings (or squares, as we denominate the space extending from one street to another), originally laid out, are in some places subdivided.

¹ It was Penn's original intention to reserve the top of the bank as a common exchange, or walk; but, on the petition of possessors of lots in front of the city, in 1684, he permitted stores to be built under the bank, expressly providing that "they should not be raised higher than four feet above the latter." "In 1690, during his residence in England, an appeal was made to the commissioners of property on the subject, who, yielding to the solicitations of the inhabitants, formally granted, in writing, liberty to build as high as they pleased, provided they left thirty feet of ground for a cartway under and along the said bank forever; when necessary, they were required to wharf out, in order to preserve the proper breadth." "The permission was gladly accepted, and the whole bank has been built upon; and not a house, as far as Pine Street, has a single foot of yard-room" (p. 23). "Southward of Pine, there is an offset of about eighty feet eastward, and the street from thence to Cedar—the southern boundary of the city—is forty-five feet wide, and called Penn Street" (p. 24).

These receptacles, which are more frequently found in the southern and north-western portions of the city proper, and still oftener in some parts of the suburbs, are occupied by the black and poorer classes of the population; by stables, and other establishments of the kind. Alleys of twenty feet wide are paved and lighted; the houses have yards and privies; but, in the smaller ones, nothing or little of this is to be found. They are not paved, and seldom cleansed; they are ill-ventilated, compactly built, in many places with small, badly-constructed frame houses, many of which are in a state of dilapidation and crowded, and present often a melancholy picture of filthy wretchedness and misery.

The wharves, or quays, which now extend several miles along the margin of the Delaware, are built of square casements of logs, filled up with earth, vessel-ballast, and stones. The surface of these wharves, as also of the street running all along the river, is paved—an improvement of the last twenty years only—while the street itself, which, from the projection of many buildings, was both irregular and inconvenient, has been, by means of funds bequeathed for that purpose by the late Stephen Girard, straightened, in many places widened, and, in the main, greatly improved. The wharves so constructed project, in many places, into the river, leaving open spaces or docks between the projections, where vessels are moored. By this arrangement greater room is obtained, and the vessels inclosed are protected from the destructive effects resulting from the action of the floating ice. But while some advantages accrue from the protection thus afforded, they are counterbalanced, whatever care may be taken, by inconveniences of a serious character in a hygienic point of view; for these recesses easily becoming receptacles of filth, and the wharves generally being elevated above the highest tides, it follows that, at low water-mark, their sides, and the half-dried surface of the docks, are exposed to the vertical rays of the sun, and become thereby the source of offensive, and at times injurious effluvia.

For a long while after the settlement of Philadelphia, the practice of city interments was exclusively followed; the graveyards used for that purpose being usually situated around their respective churches, sometimes in detached spots. In progress of time, the accumulation of dead remains has become considerable in many of the places of burial; in some of which it has been thought necessary to place stratum on stratum of earth, so as to make room for additional supplies of bodies. During the last twenty or thirty years, some of the cemeteries have been converted into open squares or promenades; others have been partially abandoned; while several establishments, of greater or less extent—some of them of considerable beauty—have been made at various distances around the city.

Until very recently, Philadelphia consisted of the city proper, having a front of a mile along the Delaware, and extending from that river to the Schuylkill, and of several suburbs or districts situated at its southern and northern extremities. These suburbs, which are continuous with the former, and divided from it and from each other by arbitrary lines only, are constructed agreeably to the same plan, and in the same manner. Thus, to the southeast, we had 1,

the District of Southwark ; 2, to the southwest, that of Moyamensing ; 3, to the northeast, the District of the Northern Liberties ; 4, to the west of this, that of Spring Garden ; and 5, to the northwest, the District of Penn Township. The 1st and 3d border on the Delaware, while the others extend to the Schuylkill. Beyond, and on the north side of the Northern Liberties, lay the District of Kensington, which, in former days, when these Liberties did not exist—or even long after they had commenced to sprout up—consisted in a separate and detached village, situate at a distance of more than a mile from the centre of the city, and separated from the adjoining part by a small stream of water. From the gradual extension of the improvements, it is now continuous with the rest, and, together with them, constitutes part of this huge consolidated city.

The surface covered by the city proper and the greater portion of the districts, has been levelled and graded, and so arranged as to be freed from almost every source of malarious exhalation—with the exception of the vicinity of Kensington, a large portion of the ground around which, and along the stream already mentioned, is, or was until lately, covered over by the tide, and presented many of the characters of a marsh.

The city proper contains but few manufactories of an injurious or offensive character—of starch, candles, or soap. There are none of glue, or of similar character—no tanyards, &c. The streets, lanes, courts, alleys, and market-houses, are cleansed by scraping, sweeping, and washing. The gutters are freed from dirt, refuse, and stagnant water, and all the dirt and vegetable and animal matter thus collected is removed, as also all kitchen offals and coal dirt, which are placed in some spot accessible to the carman. By these means, and the removal of all substances that may become a nuisance, the city is kept as clean as any other in the Union, and as most of those elsewhere. At the same time, it cannot be denied, that the regulations adopted for that object are not always so properly and strictly enforced as would be required to insure complete success.

The streets of the city run east and west, and north and south ; the former extending from the Delaware in a straight line to the Schuylkill—a distance, as we have already seen, of about two miles. The latter commence at the southern extremity of the old suburbs, and terminate at the opposite end of the northern districts. They in many cases cover a surface of several miles, and, like the former, which they intersect at right angles, are straight and regular. The city proper, which, from its being most frequently the seat of the disease to which our attention is drawn, requires a more special notice in this place, is divided into a northern and a southern portion by a wide avenue called High Street. The latter, which is one of the great business thoroughfares of the place, and in the centre of which lies the principal market-houses, is not less than a hundred feet wide. The other streets running parallel with High Street (east and west), are named from the native trees which grew in the vicinity. Beginning at High Street and going north, we have Mulberry (or, as commonly called, Arch Street, from an arch which formerly ran across it near the Delaware), Sassafras (or Race), and Vine

Streets; while, from the same starting point, and proceeding in a southern direction, we have Chestnut, Walnut, Spruce, Pine, and Cedar (or South) Streets. Besides the foregoing, or principal streets, there are a few others not included in the original plan. These run to the Schuylkill, but do not all commence at the Delaware—*i. e.* 1. Lombard, situated between Cedar and Pine; 2. Locust, north of Spruce; 3. George, between Walnut and Chestnut; Filbert, north of Market; Cherry, north of Mulberry, &c. The principal streets running north and south are twenty-two in number, and derive their name from their numerical order, counting, until recently, from each river, and proceeding towards a large avenue situate at two-thirds of the distance from the Delaware to the Schuylkill, and denominated Broad Street. Thus, we had Front Street (the first in order), Second, Third, Fourth, &c., up to Thirteenth on the Delaware side, and Eighth on the Schuylkill side. These are exclusive of the narrow street already mentioned as running under the bank of the river, and which, from its position, is denominated Water Street, and of another of limited width called Juniper, placed between Thirteenth and Broad. The streets are now numbered continuously from the Delaware to the Schuylkill.

In this enumeration of the streets of Philadelphia, I have taken no account of many others not included in the original plan, and laid out either by the State or city government, or by private individuals, some extending to a considerable distance, but generally through one or more blocks or squares.

But, although the streets of the city proper are laid out in the manner indicated, and intersect each other at right angles, there is one exception to the rule. Dock Street, originating in a way presently to be adverted to, so far from proceeding, like all the others, in a straight line, runs diagonally. Beginning at the Delaware, a little north of Spruce Street, it extends in a northwesterly and a somewhat serpentine direction through two squares across Second and Walnut Streets, and terminates in Third, a little to the south of Chestnut Street. A small branch of this street, under the name of Little Dock, extends southwesterly across Spruce above Front, and terminates in Second, a little south of the first-named street.

The streets of the districts are laid out on the same plan as those of the city; but on each side of the latter we find an exception to the straight line and rectangular course aimed at. On the south side, the Passyunk Road commences at Cedar Street, between Fourth and Fifth, and proceeds in a southwesterly direction to that part of the adjacent country called the Neck; while on the north side, the Ridge Road or Avenue, which leads to the banks of the Schuylkill, and thence far into the country, begins at the corner of Vine and Ninth, and proceeds diagonally towards the northwest. Both of these, which in their origin were, as their names indicate, common country roads, have now, from the extension of the improvements and growth of the districts, been converted—so much of them at least as is included in the latter—into streets as compactly built as those by which they are surrounded.

The streets in the city vary somewhat in point of width. High Street, as already stated, is about 100 feet wide; Broad Street is 113 feet; Mulberry,

66; Front Street, on both rivers, is 60 feet; all the rest are 50 feet, with the exception of Cherry and George Streets, which are 40, and Juniper, which is 28 feet wide. The squares or blocks in the city are, as may be presumed from the limited number of main streets and the extent of the surface covered, of large dimensions. Those from the Delaware to the Schuylkill measure from 250 to 500 feet, giving an average of 387. Those in the other direction vary from 235 to 632 feet, and give an average of about 356. These dimensions, which are confessedly too extensive, have led to the formation of those narrow streets, alleys, lanes, and courts adverted to. These may be required for the accommodation of an increasing population—especially of the poorer classes; but they are highly objectionable in a hygienic point of view. In the more thickly settled parts of the city, they have recently become, owing to the high value property has therein attained, greatly multiplied; and, from the crowded state and imperfect construction of the houses, baffle every effort at the preservation of a healthful cleanliness.

To what precedes it remains to add, that the city possesses six large open squares, five of them elegantly ornamented, and laid out and used as public promenades. One of these is situated near each angle of the city: 1, the Washington Square (southeast); 2, Franklin Square (northeast); 3, Rittenhouse Square (southwest); 4, Logan Square (northwest). The 5th, Independence Square, stands in the rear of the State and Court-houses, while the 6th, but recently improved and opened to the public, is divided into four small plots, and is situate near the centre of the city, at the junction of High and Broad Streets.

I have now presented a brief description of the city of Philadelphia—such as it exists at the present day—dwelling more particularly on those parts that have been at various times the seat of the yellow fever. But, in order that the reader may form a correct idea of the circumstances under which the various epidemics occurred, it is necessary to call his attention to the condition of the city at or about the period of each visitation; and to note the changes that have been effected since the first appearance of the disease.

Every one acquainted with the history of this country knows that the annals of the city of Philadelphia cannot be traced back beyond a period of one hundred and seventy-three years. A short time prior to its settlement in 1682, the whole surface upon which it now stands was covered by a dense forest. It was gradually cleared; but a long while elapsed before the city plot assumed its present aspect. This surface, now so level, and exhibiting so gradual a declivity from the centre of the plot to both rivers, presented a very different appearance; containing, it is true, much level ground, but also numerous hills and depressions, which in the progress of time have, by the cutting down of the former and the filling up of the latter, been completely removed. The bank along the margin of the Delaware, from Vine to Dock Streets, was high and precipitous. South of Dock Street the ground again rose suddenly, and assumed the form of a high hill—known, for a long while, by the name of Society Hill.

The plot being selected by the founder, owing in great measure to its

seeming to him "appointed for a town because of its coves, docks, springs, and lofty land," was soon after, in 1683, surveyed by Thomas Holme, the first Surveyor-General. The plan proposed by the latter was adopted, with some modifications, and followed and extended beyond the limits assigned. But, though he laid out streets in large number as high up as Broad Street, directing that in the centre there should be left a square of ten acres, "at each angle of which public buildings were to be erected; and in each quarter of the city a square of eight acres for the like uses as the Moorfield in London,"¹ the improvements were for several years limited to the river side, principally about the origin of the present Dock Street. In 1683, the year of the survey, the town, as Penn himself informs us in a letter dated 28th July, contained about eighty houses; and two years after, Robert Turner, in writing to the founder, says that "six hundred houses had been built in three years." The idea of Penn, as expressed in his instructions for settling the colony, dated September 30, 1681, was that each house should be in the middle of the breadth of the ground, so as to give place to gardens, &c., such as might "be in a green country town, which might never be burnt, and which might always be wholesome."²

The improvements were, as is well known, extremely rapid in their progress. The surface was cleared of trees; houses and stores were erected in quick succession. But, such was the extent of the plot laid out, that much time elapsed before it could be somewhat densely covered, except in the immediate vicinity of the spot originally settled, and along the banks of the Delaware.³ In 1768, when Philadelphia had become populous, and assumed the character of an important place, Nicholas Scull made a survey of the improved parts, when these were found to extend no further west than Eighth Street, and to be in a great measure restricted, in a northern and southern direction, within the limits of the city proper. Such was the case, also, at the period of the Revolution, when the whole space beyond Broad Street was thickly wooded; a condition from which it was relieved by the British troops, who, during their occupancy of the city, cut down the greater number of the trees.

But, whatever may have been, in a hygienic point of view, the advantages resulting from the plan selected by Penn, they were soon lost sight of. Houses were built more compactly than he had contemplated, so that to-

¹ Watson, i. 45, 49.

² Ibid., i. 43.

³ We learn that in 1726, forty years after the settlement, George Warner, a Friend, who died in 1810, walked from the Swedes' Church, then "far below the great town," but now in the District of Southwark, to the Blue Horse Tavern (which stood till 1828 at the southwest corner of Cedar and Ninth Streets), and saw nothing in his route but swamps and lofty forests; no houses, and abundance of wild game. From that tavern he walked to the drawbridge—at the foot of the present Dock Street—but saw no houses, and the same character of woody earth. There were but three or four houses between the drawbridge and the Swedes' Church, and those were in small clearings without inclosures. North of the bridge only two wharves. He admired the growth of the forest trees while walking up High Street, especially beyond Centre Square. (*Watson*, i. 51.)

wards the close of the last century they presented, in some places, much the same appearance, in that respect, which they do at the present day; while the blocks or squares, which had originally been made too large, began early to be cut up by alleys, lanes, and courts, which, added to the natural defects and irregularities of the unimproved portions of the city plot, towards the removal of which no efforts had, as yet, been, or indeed were for a long time made, tended to destroy the purity of the air.

The space now occupied by what is called Dock Street, together with another portion situated between the latter and Spruce, and upon which city stores have since been erected, was formerly nothing better than a miry swamp, and was given by William Penn "for the common use and service of the city, and all others, with liberty to dig docks and make harbors for ships and vessels, in all or any part of said swamp."¹ From this swamp, which was converted into a dock, arose a creek or small stream of water, which, following the course pursued by the present street, and penetrating somewhat higher up, terminated about the corner of High and Fourth Streets. A wooden drawbridge was erected at Front Street, near the mouth of the inlet, to admit crafts up. The sides of the creek were in time supported by stone walls, by which it assumed much of the character of a canal; but the small force of the current favouring the disposition of the mud suspended in the water to settle, occasioned a rapid accumulation of it. From this, as well as from the circumstance that into this reservoir were discharged the products of several tanneries, situate in the vicinity, and the offals from the gutters and other sources, it became, at low water, highly offensive to the smell, and the cause of serious mischief. It was therefore, at an early period, arched over from Third to Walnut Street. In 1784, the arch was continued, principally through the active efforts of the late Dr. Rush, as far down as Spruce Street, and the surface converted to its present use. For many years subsequently, the dock and drawbridge existed as heretofore, and were made use of. But finally the latter was removed, while the dock itself was filled up effectually, excepting always an arched culvert under the pavement, through which the water of the creek and the contents of several sewers are discharged into the river. Prior to this, and so late as 1751, that part of Front Street south of the drawbridge is represented as being impassable for want of filling up. The water rose high up Spruce Street, particularly at high tides, and covered a large surface of ground—the whole of that part being low and wet. The street was often impassable all the way from Front to Second Street, and such was its condition that, in 1747, that part was held up as a public nuisance, and petitions were presented to councils praying for a change. (*Watson*, i. 340, 1.) This ground was gradually filled in, houses were erected upon it, and, to all external appearance, no difference exists between this and other parts of the city; but, to the present day, the cellars of almost all the houses are subject to overflows to the great annoyance and injury of their inmates. In the vicinity of High and Fourth Streets,

¹ Charter of the City.

where the Dock Creek terminated, there existed a large pool filled with splatterdocks and abounding in wild ducks. So late as the year 1727, High Street, in the vicinity of Fourth, was described as a plashy place, equivalent to a *water lot or puddle*. In 1740 it was almost impassable, and it does not appear that anything effectual was attempted to change this condition of things till the year 1790.

Other streams of water, besides the Dock Creek, originally crossed the city plot; but these, in the progress of improvement, have in like manner disappeared—some at an early period, others towards the close of the last century, or even later. The principal of these streams extended to the corner of Mulberry and Tenth Streets, in a southeasterly direction, and crossing the present Washington Square (which, until a comparatively late period, was a potter's field, or cemetery for criminals and the poor), discharged its contents into what was denominated Berk's Hollow—a kind of ravine which commenced at the Square, ran across Fourth Street a little north of Walnut, and opened into the Dock Creek.¹ The ground in the whole of this vicinity was low and miry. Nor was the plot free from ponds of various sizes. Some of these existed from the origin, and were the results of the natural configuration of the soil, while others were of more recent date and of an artificial character, being the result of the formation of brick-kilns and of the raising of the streets. Some of these ponds, particularly the former, were deep, and most of them filled with stagnant water. Those in the lower or eastern part of the city were filled up and removed at a comparatively early period; but others existed, within my own recollection, in the western part of the plot. Like the former, they have mostly disappeared.

That such a state of things, which existed during the prevalence of many of the early epidemics, and to a certain extent during some of the more recent and violent ones, should have been productive of baneful effects on public health, was a result scarcely to be avoided. Hence we find a distinguished physician of the last century, who wrote in 1766, remarking, in allusion to some of the localities above described, that “when he first came into this city (in 1734), the Dock was the common sewer of filth, and was such a nuisance to the inhabitants about it, that they were obliged to use more pounds of bark than they have ounces since it has been raised and levelled.”² Nor is it less certain that the stagnant water accumulated in the puddles and ponds alluded to, contributed, in certain seasons, to the unhealthfulness of the city.

Besides these sources of morbid exhalations, there were others, both beyond the limits of the city and within its very precincts. A small stream of water, called *Pegg's Run*, passes through a portion of the Northern Liberties and Spring Garden, which, until a few years ago, was left open and unimproved. The bottom of the stream was miry, and, at low tide and in hot and drougthy weather, was often destitute of sufficient water to carry off its contents. Receiving the offals of very many slaughter-houses, tanyards, gluc, starch, dressed skin, and soap manufactories adjoining it, as well as the contents of

¹ Watson, i. p. 405, 6; 7.

² Bond's Introd. Lecture, N. A. J., iv. 270.

two culverts, of a large number of privies, and of the gutters of the numerous populous streets and alleys it crosses, it became highly offensive, and the source of noxious exhalations. This stream, which plays a conspicuous part in the history of one of the epidemics, and was correctly pronounced *the greatest nuisance in Philadelphia*, attracted finally the attention of the public and council, and has since been culverted.¹

Between the city and Kensington, the surface was long covered with stagnant water and dams; while, beyond the village, was banked land of a large extent.² In regard to these, some improvements have been made; but, in many parts, the bed is still covered at high tide, and remains muddy and marshy when the water recedes. So late as 1797, Dr. Pascalis writes: "In the Northern Liberties and the district of Southwark, there are many vacancies on the banks of the river. Owing to the periodical floods, these form large miry grounds which are never dry, but covered either with thick beds of filth from the adjoining streets or habitations, or with rubbish, old timber, &c."³

Before the surface of the city plot and of the districts had been regularly and properly graded, and sewers and culverts in sufficient number introduced, many minor sources of exhalations existed, and contributed, doubtless, at times, to the unhealthfulness of the place. The writer just cited says: "A gutter formed by the elevation of the footway, and by the gradual descent of the street on each side, seems sufficient to receive and carry down the dropping waters (from the pumps). The declivity of the street in some places, however, is worn down, and the waters stagnate, more or less, in all the gutters. During the heat of the summer, they receive the filth from the houses, and become very noxious for want of being drained; and more especially so, since they are daily renewed by the pumps."⁴ The same writer further describes the exhalations from the sinks dug to receive the water from the gutters in parts of the city deprived of sufficient declivity—and which have made way for sewers and culverts—as highly offensive (p. 99).

Nor does it appear less certain that the privies, as constructed in former days, lent additional aid to the action of morbid causes—an effect from which we are now partially, though not entirely exempt. These necessary establishments were seldom dug to a greater depth than twenty or thirty feet below the surface, and were far from being properly attended to. In their account of the epidemic of 1798 (p. 8), Condie and Folwell remark on the subject: "They are not cleaned till nearly full, which is once in ten or twelve years, and then, in many cases, it is considered easier to cover them over and dig others. After many years' fermentation, these sinks exhale a very putrid and offensive effluvia, which is abundantly emitted when the air is moist, calm, or sultry, particularly towards the end of summer, or after the action of severe heat has excited the putrefactive fermentation. The inhabitants are still so well apprised of its consequences, and know so well that the depth of their wells

¹ Jackson, *Fever of 1820*, p. 47, and Appendix.

² Barnwell, 368.

³ *Fever of 1797*, pp. 94, 95.

⁴ *Ibid.*, p. 95.

(for water) is not a sufficient preventive against this offensive exhalation, that they generally provide their necessities with an air-pipe, rising a few feet from the top; but still, under particular circumstances of the atmosphere, the ascending effluvia is frequently diffused round the houses, alleys, &c., when it is highly nauseous."¹

In one point, Philadelphians of the present day enjoy a decided advantage over their predecessors of the last century, *i. e.* in the possession of a full supply of good and wholesome water. "Our city," says a writer already quoted,² "is supplied with no running water or fountains, and but few cisterns are to be found in private houses. Wells only are dug in distributive number, and different parts of the city and suburbs. A pump is placed in each well, and water is thus easily obtained for all the wants of life." But water thus obtained, though originally good, became, as population increased, contaminated; for though the privies were not dug down, as they are mostly now, sufficiently low to reach the water, but only to the gravel bed, the water in the pump-well received the infiltration from those accumulations, as they did, also, from graveyards, until it became, finally, in some places, so offensive as to be unfit for use, even for washing purposes.³ This state of things continued until the year 1799, when the waterworks went into operation, and the Schuylkill water was distributed.

The streets, too, until somewhat later than the middle of the last century, were unpaved, and very defective in point of regulation. Dr. Franklin, to whom the city of Philadelphia is indebted for many of its early and most important improvements, was the active agent in obtaining the one in question. After showing the advantage that would result therefrom, by having a patch of pavement made in front of his own dwelling, and extending from the market-house to the footpath, he drew up a bill for paving the city generally, and brought it into the Assembly, of which he was an active member. This was about the year 1757, just before he sailed for Europe.⁴ The bill passed during his absence, and measures were taken to carry it into execution. Referring to the subject in 1766, and impressed with a conviction of the necessity of the measure, Dr. Bond pays a tribute to the wisdom of the legislature in framing the salutary law in question, and to the indefatigable industry and skill of the commissioners in executing it, "whereby they have contributed so much to the healthfulness of the inhabitants."⁵

But while attention was thus called to the necessity, on the score of public health and private convenience, of paving and otherwise improving the streets, nothing, for a very long while—and, indeed, until within a very few years—was done to place the wharves and the adjoining streets in a proper and safe condition. Constructed in the manner described, they were left unpaved, and thereby, at certain times, became miry and almost impassable; while the avenue along the river was irregular and compact, the buildings, for the most part, old and dilapidated, and crowded with a low class of the population, and with

¹ Condie and Folwell, p. 8.

² Pascalis, p. 95.

³ Condie and Folwell, p. 8.

⁴ Life, p. 169.

⁵ North Am. Med. and Surg. Journal, iv. p. 270.

stores dirty and badly kept. Filth was allowed to accumulate in the docks or interstices between the projecting piles or slips, as well, also, as upon the wharves themselves. Let the following description of a portion of these localities, as they existed in 1820, serve for a tolerably accurate picture of the whole at that and antecedent periods: "Hodge's Dock (near Sassafras Street).—This dock has been neglected for some years, and has gradually been filled up. At low water, it is at present uncovered nearly its whole extent, and a large mass of mud, of animal and vegetable remains, is thus exposed to the action of the sun and air. Two culverts, or tunnels, into which empty the privies of the range of frame buildings on the north of the wharf, and those of some of the houses in Water Street, to the north of it, discharge their contents into the dock; and the inhabitants of this part of Water Street, few of whose dwellings have privies, are accustomed to throw into it the contents of the buckets, &c., which are employed as substitutes for those indispensable conveniences."¹ "The wharf near to the dock is higher than where distant from it, so that the water cannot drain from the wharf, but stagnates and evaporates on it." The people residing along it, "some of whom were washerwomen, were in the habit of throwing all their kitchen water, offal matters, and soapsuds" "on the wharf, and on a pile of plaster of Paris that had been there for two or three years. The pile of plaster had in this manner become a receptacle of filth, which filled up its interstices, and the wharf was kept in a foul and offensive condition" (p. 36). Of another portion of these localities, the same author remarks: "Immediately above Walnut Street, large quantities of molasses had been landed, the hogsheads of which were in a very bad condition, and, in consequence, had leaked considerably. From the frequent showers that fell, the wharves were moist and soft, and the molasses became worked up, by the constant passing and repassing of carts, drays, and people, with the mud, which is a compost of various putrefiable and fermentable matters" (p. 41). It is easy to conceive that when wharves thus constructed and circumstanced become exposed to the fervid beams of our summer sun, the putrefactive fermentation will easily be occasioned, and noisome and deleterious effluvia produced.

In view of this condition of the localities described, and of those portions of the city resorted to by the colored and poorer classes of the population—to which I have before called attention—we cannot but admit the propriety of the censure of early writers on this country, one of whom, the oft-quoted Volney, does not hesitate to place Philadelphia, in point of cleanliness, beneath other cities of far inferior pretensions. "The Americans," he says, "boast of their cleanliness; but I can aver that the wharves of New York and Philadelphia, as well as certain portions of the suburbs, surpass, in public and private filthiness, all I have witnessed in Turkey, where the air possesses the advantage of being of a salutary dryness."²

That a beneficial change has been effected since the days of this highly-intelligent traveller, so far, particularly, as the morbid influences are con-

¹ S. Jackson, *Fever of 1820*, 36, 37.

² *Climate des Etats-Unis*, p. 347.

cerned, which have usually been found associated with the production and dissemination of the form of disease which constitutes the subject of our present inquiries, no one among us will refuse to bear witness. It is much to be able to say, in connection with that fact, that the city plot has been properly graded, drained, and paved; that creeks have been culverted; that pools, ponds, swamps—formerly the source of much insalubrity—have been filled in, and their surfaces variously improved; that a full supply of pure and wholesome water has been obtained; and that the dock has been filled in. It is much, too, to notice that the hygienic condition of the river side has been highly ameliorated; that the wharves have been paved and rendered otherwise less objectionable; that Delaware Avenue has been straightened, and, together with Water and the adjoining streets, has been greatly improved; and that better police regulations have been introduced. All this has been subject of observation among us, and no one has failed to perceive that, by these various means, and the gradual spread of improvement, malarial fevers have been chased beyond the limits of the city north, south, and west, and the sources of noxious effluvia, on the east front, greatly lessened. In a word, while the city generally has been, in several respects, cleared of many of the conditions of locality more or less connected with the causation of fevers of telluric or malarial origin, or which add force to epidemic and other maladies, those parts of it which exhibit the peculiar local features usually and everywhere recognized as connected with the origin of yellow fever, and where that disease, at each of its epidemic visitations, has broken out and spread, have been advantageously modified.

But, while admitting all this, no one acquainted with the condition of the city, or who has perused the preceding statements, will refuse to confess that much yet remains to be done ere Philadelphia can justly deserve the reputation for unsurpassed cleanliness and neatness in all its parts, so often claimed in its behalf; that the facilities of ablution, which the abundant supply of water furnishes, are much more neglected than is conducive to public comfort and health; and that so far as concerns the obscurer parts of the city, alleys, lanes, courts, and especially the wharves, docks, the outlets of sewers, &c., much remains to be done by the guardians of public health.¹ Morbid influences which, in other cities or regions, subject the residents and visitors to febrile diseases, and especially to that form of them which constitutes the main object of our inquiries, exist in our midst, and existed to a greater extent at an earlier period of our history. Nor could it be otherwise; for the effect is the result of circumstances over which we have little or no control, and which could not be removed but at an expense which no city government would be willing to incur, and at sacrifices to which it would be vain to expect any community to submit. They depend on the natural, uneven, and irregular condition of the city plot; on the vicinage of the river side; the peculiar quality, natural and artificial, of the soil, in many parts at least of

¹ See Report of Joint Special Committees of Select and Common Councils; Report on Public Hygiene in *Trans. of Am. Med. Assoc.* for 1849.

its surface; the compact construction of the houses; and the narrowness, imperfect ventilation, and filthiness of the streets in certain sections of the city. They arise also from the numerous courts and alleys, many of which are closed at one end, unprovided with a sufficiency of water, imperfectly drained, often unpaved, to say nothing of the piggeries and other nuisances they contain; the denseness and squalid character of the population of these localities; the defective mode of construction, and the physical peculiarities of wharves, built of perishable timber, and filled up with animal and vegetable remains and rubbish of various kinds; as well as from the condition of the docks, and the proximity of the shipping. Objectionable localities—though more numerous, perhaps, formerly than they are at present—may be found in almost every district, even in the very heart of the city, but abound more in some portions of it than others. Hear what is said on the subject in the report of the Sanitary Committee of the Board of Health:—

“It should not be overlooked, that there exists a limited district in one of our suburbs, a locality that is abundantly fruitful in originating or propagating disease, and where at all seasons, winter and summer, spring and autumn, we anticipate its irruption in some frightful or protean form. In this almost isolated neighbourhood we find an excess of a vagrant population, half fed and half clothed, crowded together in almost untenable houses, and in open and humid cellars located in narrow streets and narrow alleys, and pent-up courts, badly ventilated and badly lighted. In this forbidding district, we could enumerate many fruitful causes for disease operating upon the half-famished and bloated bodies of a depraved and mixed population, whose constitutions have been undermined through the ravages of intemperance and exposure, the accumulation of filth, and impure food and air, accompanied by the far more ruinous effects of a life of moral shame and degradation, become the fit receptacles, and afford the materials for the growth and reproduction of those morbid agents which constitute disease.”¹ “In the city proper, and along the west side of Water Street, between Vine and Arch Streets, may be found dwellings very illy constructed for a free circulation of air, without yards, and erected under a towering bank, where but little air and sun reach the lower apartments. These houses are mostly occupied by the lower classes; and in many of them may be found a family in every story.” Many of these houses—often the seat of yellow fever irruptions—as in those on the eastern side of Front Street, have no privies.

CLIMATE.—While the city of Philadelphia exhibits, in several of its localities, many of the peculiarities which, in other places subject to the yellow fever in its endemic, epidemic, or sporadic aspects, are found associated with the origin or diffusion of the disease, and which in all probability play an essential part in its production, it is found to possess a climate not dissimilar, during certain seasons at least, to that of regions which constitute the legitimate home of the fever.

¹ Report of Joint Committee, p. 36.

The climate of Philadelphia, like that of the surrounding country, is one of extremes; the cold of winter, as well as the heat of summer, being almost invariably considerable, and at times of very great intensity. But, though thus noticing these extremes of temperature, as well as those of moisture or dryness exhibited in the atmosphere, I shall more particularly point out, in accordance with the true import of the science of climatology, the averages in both divisions of that important subject.

It has been stated that in the whole of the 40th degree on the Atlantic coast of America, the four seasons attributed to regions in the temperate zone are well defined. Spring is recognized in March, April, and May, by a gradual increase of temperature. Summer, evinced by an extreme accession of heat, follows in June, July, and August. Autumn is marked in September, October, and November, by a gradual decrease of temperature. Winter, which follows in December, January, and February, is characterized by, sometimes intense, frost; by snow; and at all times by a considerable depression of the thermometer.

Without inquiring how far this statement will apply to other localities included within the parallel in question, experience leaves no doubt of the fact that, in Philadelphia, and Pennsylvania generally, the four seasons are not all usually found to be as equally and distinctly defined as might be thereby inferred. So far from this, it is noticed that though the winter, summer, and autumn are tolerably well marked, the spring can scarcely be said to exist as a separate season. The severe cold of winter is quickly succeeded by the equally severe heat of summer, the season being tardy in proportion to the severity of the preceding winter, so that the mild weather which characterizes that season in parallel countries seldom endures more than a month, and this not always consecutively, but in short detached portions. In March, the weather is usually stormy, variable, and cold. In the month following, and sometimes as late as the beginning of May, the atmosphere is moist, often unpleasantly cold, and characterized by a condition which we denominate *rawness*. Snow often falls in that season, and has occasionally been known to show itself as late as the first week of May, the latter portion of which month is mild, but at times extremely warm. In June, the heat, which is occasionally severe and oppressive, is usually temperate; the sky being serene, and the verdure wide-spread and delightful. This month, indeed, is the only one resembling a spring month of the south of Europe. Sometimes during this month, and the succeeding part of spring, the weather is cloudy and damp, and attended with heavy showers. Towards the close of June, and during the whole of July, which is usually the hottest month of the year, the thermometer rises high, the temperature being intense and oppressive, and the weather dry and drougthy. In August, the extreme heat continues with little change during the day, but decreases sensibly after sundown, and at night becomes moderate, while the atmosphere loses its dryness by the occurrence of frequent showers. The early part of September presents, in its temperature, the same characteristics as the latter end of August; warm, and sometimes hot days, being followed by cool and pleasant

evenings and nights. But, from about the 10th or 15th of that month, when an agreeable change occurs in the temperature and weather generally, autumn, decidedly the finest and most agreeable season of the year on this side of the Atlantic, fairly sets in. "During the latter part of September, and quite the whole of October, the weather is most commonly serene, and the temperature usually remains in that condition which neither produces a sensation of unpleasant warmth, nor, to the healthy, sufficient coldness to render fires necessary for their comfort. This period has been declared, by Americans of observation, who have spent years in Italy and southern France, to excel the most pleasant seasons of those countries, so famed for the beauties of their climates. Slight white frosts are noticed on low grounds in the latter half of September, and general white frosts caused by a morning temperature of 42° and 40° appearing in the first ten days of October."¹

The rain now commences, sometimes in heavy, at others in gentle showers, which are often repeated during several successive weeks, and interspersed with fine, cold, and serene days. From the close of this month, or the beginning of November, the cold gradually increases, attended very generally with fine, clear, dry weather; and frost and snow show themselves. In November, however—if it has not done so in the latter part of October—rain succeeds to the fine weather described, and proves the harbinger of winter. The latter now opens with more or less severity; but, though the month of December is characterized by a great depression of the thermometer, it seldom happens that intense cold sets in before the 20th or 25th of that month. In January, the winter attains its culminating point; that month presenting, generally, the lowest mean temperature, and being characterized by a more constant succession of often intensely cold weather, with its necessary concomitants, ice, snow, &c. From the close of this month the season may be said to decline; for though, in February, the thermometer occasionally falls considerably—sometimes as low as it had done at any preceding time—such spells are not of very frequent occurrence, and besides, are of shorter duration, the temperature becoming gradually more moderate, and less uniformly cold.

A review of the characteristic peculiarities of the seasons in or about Philadelphia, will show that, notwithstanding the extreme heat we suffer during the summer, coldness is the predominant feature of our climate, as there are not, in all the year, more than four months—one-third of the time—in which the weather is agreeable without a fire.

Enough has been said to show that the spring, from the short duration of pleasant and moderate temperature, the uncertainty of the weather, and other causes specified, is far from being an agreeable season in this vicinity; and I have dwelt on the contrary character of our autumn. Before closing the subject, it may not be improper to remark that, notwithstanding the length and severity of the winter—notwithstanding the quantity of snow that falls and accumulates to the depth not of inches only, but at times of feet—notwithstanding the intensity of the cold and the solidity and duration of the ice,

¹ Sexton, Am. J., v. 51.

the season is far superior in point of agreeableness to the succeeding one. During the greater part of it the weather is, at least in ordinary years, clear, dry, bracing, and elastic. Hence, as we shall presently see, it is, in point of healthfulness—judging from the mortality—the most advantageous section of the year.

Very different, in many respects, is the summer season, one of the most important in reference to the subject more particularly before us. Dr. Rush remarks (pp. 15, 16) that the air, when dry, in Pennsylvania, has a peculiar elasticity, which renders the heat less insupportable than the same degree is in moist countries, and that it is in those cases only when summer showers are not succeeded by northwest winds, that the heat of the air becomes oppressive and distressing from being combined with moisture. That this is in the main correct there can be little doubt—so far, especially, as the country adjacent, to say nothing of the rest of the State, is concerned. Nothing can exceed, in point of agreeableness, the atmospheric conditions consequent on the change mentioned. But the northwest wind does not invariably succeed to these summer showers, and when it does not, we have a sultry and humid state of air, which is far from being pleasant. Besides which, there are in the adjacent country, as also within the limits of the city, other conditions of atmosphere, which, in conjunction with that already mentioned, render the summer season more disagreeable with us than it is in many other countries equally warm; while, from its position and mode of construction, the city itself suffers more in that respect than other cities. Well could Volney, long ago, dwell on the disagreeableness of the heat in Philadelphia (i. 144). It approximates the climate, during several months of the year, to that of tropical regions, and hence, aided by a frequent absence of refreshing breezes and by long-continued droughts, entails upon the city some of the morbid influences from which those regions suffer, and to which they owe, among other afflictions, the poor privilege of being the principal, and almost annual birthplace of the disease before us.

As regards the range of atmospheric heat observed among us, we may safely affirm, that it rarely happens that more than twenty or thirty days present themselves in summer or winter in which the mercury in Fahrenheit's thermometer attains a height of more than 80° in the former, or falls below 30° in the latter season.¹ From a review of the results of observations made during a long succession of years—from 1758 to 1848 inclusive—it will be found that the annual averages varied from 48° (1836) to 56° (1819);² giving a mean temperature throughout the year, for the whole series, of 53° —this being the temperature of the deepest well-water.

It has already been mentioned that the climate of this place is one of extremes. To justify the statement, it will be necessary to point out not only the mean temperature of the whole year of the several seasons, and of each particular month, but the highest and lowest points to which the thermometer

¹ Rush, 81.

² Summary of College of Phys., i. 340, ii. 261; Hewson, Am. Phil. Tr., vi. 395-8; Forry, Climate of United States, p. 10.

reaches. A series of observations to that effect enables us to discover that, though some years have occurred when the greatest heat has not exceeded 83° (1768), it more usually happens that from the latter part of June to the beginning of August—the usual period of the greatest heat—the thermometer ranges, during a succession of days, from 90° to 96° —and in some seasons reaches as high as 100° ; whilst in the winter months, it descends often as low as zero, and even, at times, several degrees (varying from 3 to 7) below that point. These, however, are not to be assumed as samples of our ordinary temperature, the thermometer more usually ranging, during the hottest part of summer, somewhere about the 85th or 86th degree, and seldom descending, in the coldest weather—except, perhaps, occasionally for a day or so—below the fifth degree above zero. From this it would follow that, in ordinary years, the extreme difference of temperature between the summer and winter seasons, amounts to 80° . But, however this may hold as a general rule, the difference, taking into account the extremes noticed above, is often much greater.

The following table, compiled from Forry and other sources, will present a tolerable idea of the averages at the different periods of the year:—

	1771, 1772, 1775.	1798, 1799, 1800.	1811 to 1820.	1822, 1823, 1824.	1835 to 1840.	1841 to 1848.	Average of 33 years.
Spring	50.88	52.44	49.6	52.11	50.8	52.3	51.27
Summer	71.62	75.03	72.6	76.16	71.6	72.1	72.68
Autumn	54.32	55.21	55.3	59.10	52.8	55.3	55.33
Winter	34.06	33.02	32	32.23	30.8	34.5	32.76

In the series of years already referred to, extending from 1758 to 1848, the extreme maximum was 98° (1843), and the extreme minimum 3° below zero (1775), the difference being 102° ; the highest point reaching 55° above the mean annual heat, and the lowest 56° below the same. In the same series, the annual range varied from 93° (1775) to 69° , being thirty times in 49 years at or above 80° ; and presenting, for the whole period, a mean of $81^{\circ} 1'$. The following table of the mean temperature of each month during the years 1852, 1853, 1854, I owe to the courtesy of Prof. Kirkpatrick, of the High School, whose accuracy in researches of this kind is well known.

	1852.	1853.	1854.	Av. of the 3 yrs.
January	25.82	32.02	30.42	29.42
February	33.05	35.99	33.42	34.15
March	43.09	41.80	42.22	42.37
April	47.26	52.15	50.08	49.83
May	64.10	63.10	64.25	63.82
June	71.60	73.58	71.63	72.27
July	76.86	75.11	79.11	77.03
August	72.67	74.85	75.49	74.34
September	65.89	68.32	70.02	68.08
October	58.27	53.21	59.15	56.87
November	42.15	47.13	46.13	45.14
December	40.37	33.81	31.26	35.15
Mean temperature	53.429	54.256	54.432	54.039

	1852.	1853.	1854.	Average.
WINTER—Dec., Jan., Feb. . . .		36.127	36.55	for 2 yrs. 36.338
SPRING—March, April, May . .	51.45	52.35	55.517	for 3 yrs. 53.106
SUMMER—June, July, August . .	71.783	74.513	75.403	“ 73.899
AUTUMN—Sept., Oct., Nov. . . .	55.447	56.22	52.47	“ 54.712

It will be found, on examining the whole number of observations from 1758 to 1854, that of the twelve months, the one which, by its mean amount of heat, expresses the nearest equivalent to the mean annual temperature, is October; a result corresponding to that pointed out by Humboldt in reference to modified climates generally. The months which exceed the annual mean are May, June, July, August and September; while those that fall below are January, February, March, April, November, and December.

Dr. Rush, in his *Essay on the Climate of Pennsylvania*, says: “The warmest parts of the day in summer are at two o’clock in ordinary, and at three o’clock in the afternoon in extremely warm weather. From these hours the heat gradually diminishes till the ensuing morning. The coolest part of the four and twenty hours is at the break of day. After the warmest days, the evenings are generally agreeable, and often delightful. The higher the mercury rises in the daytime, the lower it falls the succeeding night. The mercury at 80° generally falls to 68°; while it descends, when at 60°, only to 56°. This disproportion between the temperature of the day and night in summer, is always greatest in the month of August. The dews, at this time, are heavy in proportion to the coolness of the evening; they are sometimes so considerable as to wet the clothes; and there are instances in which marsh meadows have been supplied with their usual waters from no other source than the dews which have fallen in this month, or in the first weeks of September.”¹

The preceding remarks have reference to the country generally. It is proper to say, therefore, that the city of Philadelphia has, to a certain extent, a climate of its own, so far as temperature is concerned, the thermometer ranging, in the shade, somewhat higher than in open localities of the vicinage. From what precedes, it results that while in regard to the amount of heat it differs little from the Atlantic and other cities of our Middle States, it is found on comparison to equal, in its mean summer temperature, the least favoured of our southern cities, and is little inferior in mean results, and superior as to extremes, to the coast of South America, the West Indies, and even the coast of Africa. In a word, while our winters are almost Siberian in character, our summers are, as already stated, truly tropical, and capable, therefore, when aided by the conditions of locality already men-

¹ Med. Inq., ii. 9.

tioned, and by other morbid agencies presently to be adverted to, of giving rise to disorders peculiar to hot climates.

The barometer exhibits a mean elevation of about 30° ; the variations being very inconsiderable in the greatest changes of the weather. Dr. Rush (ii. p. 16) states, on the authority of Mr. Rittenhouse, that the alterations in the height of the mercury do not *precede*, but always *succeed* changes in the weather. Later observations have shown that this remark, though often correct, is not invariably so. "It falls with the south and southwest, and rises with the north and northwest winds." Observations made during fifteen years, from 1788 to 1803 inclusive; in 1805, and from 1841 to 1848 inclusive, show that the mean annual pressure was 29.988; the extremes being 30.18 (1805) and 29.86 (1799). The highest month was December, and the lowest February. The former (30.618) was .620 above the mean of the year; and the latter .018 below; the range between them being .718. Of the several months, the one exhibiting a pressure which approaches nearest to the annual mean is June, being .007 below it. Of the whole series, April, September, October, November, and December, rise above the annual mean; while January, February, March, May, June, July, and August, present a lower result.

The climate of Philadelphia, like that of other parts of Pennsylvania, differs considerably from that of England, and some other localities of the European coast, in point of the hygrometrical character of the atmosphere. Being under the influence most commonly of land, instead of sea winds, as occurs there, our atmosphere contains a less amount of moisture. (*Rush.*) Compared, however, with the atmosphere of some cities of Europe, the difference, if it exist at all, would be, even were Dr. Rush's observations well founded, less striking; for while, for example, the yearly quantity of rain averages at Rome 32 inches, at Florence 37, at Naples 34, at Padua 36, at Sienna 38, and at Venice 42,¹ the amount here, according to that eminent physician, one year in another, varies from 24 to 36 inches, the average being 30.² But more recent observations lead to the conclusion that the average quantity usually exceeds that amount. The four years, 1799, 1800, 1801, and 1805, gave an amount of $42\frac{3}{4}$, 39, $40\frac{6}{8}$, and 42 inches, the average being 41 inches.³ In 28 years—from 1810 to 1837, inclusive—the whole quantity that fell amounted to $1,035\frac{742}{1000}$ inches; and presented an annual mean of $36\frac{991}{1000}$ inches;⁴ the smallest quantity which fell in one year being $23\frac{359}{1000}$ (1819), and the largest $48\frac{560}{1000}$ (1833). From 1838 to 1841, inclusive, the average quantity of rain and melted snow amounted to 47.80.⁵ From 1841 to 1848, inclusive, we have an average of $42\frac{412}{1000}$ inches; and 1850 gave 59.54 inches;⁶ the average in 44 years being 38 inches. According to the observations of Prof. Kirkpatrick, the quantity in 1852 amounted to 46.20; in 1853 to 42.06; and in 1854 to 45.23; the average for the three years being 43.47. Observations made and recorded by Dr. John Conrad, at the Penn-

¹ Volney, i. 259; Carriere, 173, 362.

² Rush, ii. p. 16.

³ Cox's Museum, i.; Mease, Picture of Phil., 43.

⁴ Amer. Alm. 1839, p. 139.

⁵ Am. Alm. 1843, p. 69.

⁶ Sum. of the Col. of Phys., i. 2, 4.

sylvania Hospital, show that the annual average in a series of 25 years was 43.76.¹

This places Philadelphia on the same footing as most of the favoured cities of Europe, and higher than others, and many of those of this country; but, as will be seen in a subsequent chapter, much below, on the point in question, other parts of this country, the West India Islands, some parts of the East Indies, the coast of Africa, &c.

It may be added, in illustration of the variableness of our climate, that the summer and autumnal seasons are sometimes characterized by an excess and at other times by a deficiency in the quantity of rain. Droughts of months' duration are not uncommonly noticed, or shorter spells of dry weather, alternated with heavy showers or torrents, sometimes without, though generally with thunder.² At other times, prolonged spells of rain, extending to a week or even to near a fortnight, and then, after intermitting with short periods of clear and dry weather, returning and continuing for some time longer, deluge the earth and injure the crops. At other times, again, rain falls in greater or less abundance during six, twelve, or twenty-four hours, and is followed by a few hours or a day or two of fine weather. This again is succeeded by rain, and the alternation continues for some time.

The greatest quantity of rain sometimes falls in April, May, June, July, and August, or in December, and even in January. These last two occurrences, however, are rare; the former are the most frequent—the spring or summer being often the wettest season of the year. A series of observations, covering a period of eight consecutive years, 1841 to 1848, shows that, during the three months of January, February, and March, rain fell to an amount varying from 6.21 inches to 13.78, with an average of 10.36. In April, May, and June, the amount varied from 5.29 to 14.55, with an average of 9.78. July, August, and September exhibited a variation of from 6.80 to 18.74, with an average of 12.58; while the amount in October, November, and December, varied from 8.35 to 13.31, with an average of 10.90. The average of the whole year being 43.62, and that of each of the four quarters 10.15.³ From the records of the rain-gauge kept at the Health Office, in a central part of the city, from March 1820, to February 1827, inclusive, the three months of winter averaged 8.15; spring, 8.29; summer, 9.54; and autumn, 10.54.⁴

Another series of observations made at the Pennsylvania Hospital, already referred to, gives for a period of twenty-five years the following averages:—

January 3.23	} 9.78	April 3.26	} 10.70	July 4.29	} 11.93	October 3.30	} 10.76
February 2.83		May 3.53		August 4.34		Nov. 3.53	
March 3.72		June 3.91		Sept. 3.30		Dec. 3.93	

During the aforesaid period of eight years, the number of rainy days varied from 74 to 104; the whole amount being 724, with an average each year of 90.5. The number of cloudy days varied from 58 to 127, with a total

¹ Sum. of the Col. of Phys., N. S., ii. p. 168.

² Rush, ii. 16, 17.

³ Sum. of the Col. of Phys., i. and ii.

⁴ Emerson, Am. Med. J., i. p. 120.

amount of 661, and an annual average of 82.5. The clear days varied from 35 to 63; the whole number being 409, and the average 51.1; and the days in which the weather was variable, but clear during the greater or some portion of the time, varied from 191 to 266—the total being 1,839, and the average 229.¹ Our city has evidently, in that respect, the advantage over many localities enjoying a great reputation for beauty of climate. Thus, while clear, or partially clear days, constitute more than two-thirds of the whole number throughout the year, at Naples they do not exceed the one-fifth or one-fourth of the whole. The mean number of rainy days at Rome and Florence is 114; at Sienna 104.

The dew-point, in 1844, varied from 20 (January) to 62 (July); the average for each month of the year (October and November excepted) being $36^{\circ}.4$. In 1845, it varied from 25 (December) to 62 (July); the average of the year being $42^{\circ}.8$. In 1846, the variation was from 21 (February) to 65 (July and August); with a mean point for each month of $43^{\circ}.9$. In 1847, the point varied from 24 (January) to 64 (July); and the mean amounted to $41^{\circ}.3$. In 1848, March presented an average of $25^{\circ}.25$, while the dew-point in July reached $67^{\circ}.05$; the mean of each of the twelve months being $42^{\circ}.16$, and that of each of the five years $41^{\circ}.51$. In 1852, the average of the year was $43^{\circ}.72$, ranging from $30^{\circ}.50$ (Jan.) to $55^{\circ}.90$ (July). In 1853, the average was $43^{\circ}.71$, ranging from $29^{\circ}.32$ to $59^{\circ}.31$ (August). In 1854, the average being $43^{\circ}.90$, the range was from $27^{\circ}.96$ (Dec.) to $59^{\circ}.22$ (July). The spring months, March, April, May, presented averages, in 1852, of $40^{\circ}.50$; 1853, $40^{\circ}.75$, and 1854, 41° . Summer (June, July, August) gave, in 1852, $54^{\circ}.03$; 1853, $57^{\circ}.32$, and 1854, $56^{\circ}.91$. Autumn (Sept., Oct., Nov.) gave, in 1852, $43^{\circ}.63$; 1853, $45^{\circ}.44$, and 1854, $47^{\circ}.05$. The average point in the winter of 1853 (Dec., Jan., Feb.), was $34^{\circ}.20$; and the same months, in 1854, gave an average of $31^{\circ}.56$. The dew-point, as may be presumed, is much higher during the warmer than the colder months of the year. It usually commences to rise about March or April; continues to rise until it attains its maximum in July and August, and then again descends. Nor is it less worthy of observation, that the daily range of the dew-point in Philadelphia—as, indeed, in other parts of the United States—is considerable; amounting, at times, to more than forty degrees in twenty-four hours. Finally, the dew-point here and in other portions of these latitudes, is usually many degrees below the temperature of the atmosphere. As Dr. Lee remarks of our climate in general, it is but rare, indeed, that both temperature and dew-point coincide. Hence it is that we are but seldom troubled with that kind of weather which has received the denomination of close, sultry, and muggy, and is the natural result of that coincidence.

POPULATION.—The population of Philadelphia, including the various suburbs and districts within the circuit of the bills of mortality, amounted, according to the census of 1840, to 236,791; that of the whole county being

¹ Sum. of the Col. of Phys. of Philad., i. and ii.

258,037. In 1850, the population of the former reached 360,408, and of the whole county 408,862. Very different, however, was the state of things in this respect at the epoch of the various epidemics that have prevailed in the city—especially those of early times; and as it would be impossible to form an estimate of the mortality occasioned by such epidemics without a knowledge of the number of people placed within the sphere of their influence, it may be proper to present a statement of the progressive rise of the population from the settlement of the city to the period above mentioned.

In the year 1683, the number of dwelling-houses was estimated at only 80,¹ which, if we compute the inmates of each at about 6.31, gives a population of 504. Seventeen years after, in 1700, the houses had so increased as to amount to 700, with an estimated population, based on the above calculation, of 4,417.²

From that year to about the middle of the century, we have no information regarding the number of either houses or inhabitants; and even from the latter period to 1790, when, I believe, the first census was taken, our knowledge is far from being as definite and satisfactory as might be desired—the conclusions recorded being more approximative and arbitrary than founded on positive data. The following table, constructed on the information we possess, will enable the reader to form a tolerably correct view of the subject, covering as it does, in conjunction with what precedes, the several periods at which the fever appeared.

Years.	Houses.	Population.	Years.	Houses.	Population.
1683	80	504	1793		50,000
1700	700	4,417	1798	10,000	60,000
1742-5		12,000	1800		70,287
1749	2,076 ³	13,099	1805	13,461	84,878
1753	2,300 ³	14,563	1810	15,814	96,664
1760	2,960 ³	18,756	1820		121,210
1769	4,474 ³	28,042	1830		167,118
1776	5,460 ³		1840		226,693
1783	6,000		1850		360,408
1790	6,651	44,996			

The city proper, exclusive of the districts, contained, in 1790, 28,522 inhabitants; in 1800, 41,220; in 1810, 53,722; in 1820, 63,802; in 1830, 80,458; in 1840, 93,608; and in 1850, 121,381.

This population, thus accumulated, is of a mixed kind, composed, in great measure, of natives of the soil, but containing a very strong admixture of

¹ Proud., Hist. of Pennsylvania, i. p. 203.

² The number of houses erected upon the plot of the city, exclusive of the public buildings, stores, workhouses, &c., in December, 1769, were 3,318; those of the northern suburbs, 553; and of the southern suburbs, 603; in all, 4,474 dwelling-houses, which, at the most moderate computation, being multiplied by 6, gives 26,844 inhabitants; but they were supposed to average nearer 7 to a family, which makes 31,318 in the city and suburbs of Philadelphia; at that time, it was not above eighty-seven years old.—*Proud.*, ii. 277.

³ Robinson's Directory, quoted by Mease, ii. p. 5.

foreigners, principally Irish and Germans, together with some French, and a few Italians and Spaniards. The whites far exceed the blacks. The number of these, in 1790, amounted to little over 2,000. Thirty years after, in 1820, they reached to 11,384, or 1 in 10.63; in 1830, to 14,642, or 1 in 11.5; in 1840, to 18,456, or 1 in 12.36; while the census of 1850 (p. 179) gives us a total of 19,761 for the county, being in the proportion of 1 in 20.69. In the city proper, the population of which was at the last mentioned period 121,375, their number was 10,736, or 1 in 10.36.

The majority of the people may be, and I have little doubt are, descended from an Anglo-Saxon stock; but so great has been the influx of foreigners, since the establishment of the city, that a vast proportion of the present inhabitants are the offspring of these and of intermarriages. The intermingling of races here, as in most of our cities, is very great, and extends even, though on a limited scale, to a mixture of the Caucasian with the African. In former days, these mixtures were of less frequent occurrence, and the proportion of foreigners was not so great as it has become during the last thirty or forty years; hence, the early epidemics of yellow fever, which, as we shall see, were the most violent, exercised their influence principally—and, on some occasions, exclusively—on individuals of English descent, or emigrants from Great Britain.

The negroes who inhabit the city and districts are in large majority natives of the place. In most instances, their parents and ancestors, for several generations back, were so likewise. A portion, however, are derived from the nearer slave States, a few from more southern portions of the country, and a fewer still from the West Indies, and even from Africa. In the middle, and even in the latter part of the last century, and in the first quarter of the present, the proportion of the native blacks was more limited than it is now. This class of the population was then principally made up by recent importation from other States, or foreign colonies, and received, at the period of the revolution in the French West India Islands, a considerable accession to their numbers from immigration from several of these, though principally from St. Domingo.

Many of the inhabitants are in affluent, and many more in easy circumstances; and such, to a certain extent, has been the case, from an early period of the colonial existence of the country. But Philadelphia, like every other city on the surface of the globe, contained then, and contains now, in a much larger proportion than it did in early days—though less so than many places of similar kind—a large amount of poor; many of whom, in point of wretchedness and immorality, will compare with the dregs of society in other cities of our country, and even in foreign lands. With some exceptions, they are accumulated within the alleys, courts, and lanes in the southern and western parts of the city, and in various portions of the districts.

Basing our data on the bills of mortality, as well as on the private reports of individual practitioners, on my own personal observations, and on information derived from various sources, respecting the character and issue of each particular form of disease, we may, without fear of error, infer that, notwithstand-

ing the local peculiarities of the city plot, and the extremes of heat and cold, which, as has been seen, characterize our climate, the variableness of the latter in regard to the thermometrical and hygrometrical conditions of the atmosphere, and the rapidity, frequency, and extent of the changes which occur in these conditions, Philadelphia is far from being amenable to the charge of unhealthiness. Experience, indeed, shows that, taking all things into consideration, it may be regarded, in comparison with other cities of equal size, as a healthy city; for facts, easily collected, will bear me out in the opinion, that such of its inhabitants as are properly regardful of the true precepts of hygiene in point of clothing, mode of living, and habits, fare no worse than those of similar places on this and the other side of the Atlantic; escaping most of the acute diseases occasioned by sudden modifications in the sensible qualities of the atmosphere; being no more prone than individuals elsewhere to complaints arising from ordinary morbid causes, and being at the same time spared from some incident to large concentrations of population.

Be this, however, as it may, it has been remarked, with great propriety, that as we have the climates, so we have the health and acute diseases of countries of opposite geographical positions. Hence, our winter and spring are prolific in diseases commonly encountered in northern latitudes—inflammation of the serous and parenchymatous structures, tubercular and other forms of pulmonary complaints—croup, catarrh—as also, under particular contingencies, typhoid and typhous fevers. At the same time, the elevation of temperature, the excess of rain and droughts, as well as the torpid and stagnant condition of the atmosphere of summer, which, as we have seen, impart a tropical character to our climate in that season, assimilate our diseases, in some degree, to those of the West Indies, or, indeed, of hot regions generally. The heat not unfrequently proves suddenly fatal to individuals exposed to the direct rays of the sun; and, either alone or combined with other morbid agencies incident to that period of the year—humidity, malaria and other impurities—becomes a fruitful cause of cholera infantum, cholera morbus, diarrhoea, dysentery, and hepatic derangements. From the same circumstance arises a languor in the system which disqualifies those who remain in the place from resisting the influence of febrile poisons, the materials of which, as we have seen, are abundantly encountered here. Hence, remittent bilious and other fevers of various grades, bilious pleurisies, and other diseases of kindred nature, prevail to a certain extent in that season, but become particularly rife in the autumn, when they often assume the character of a true endemic, and sometimes spread with epidemic violence.

In this short summary of the diseases ordinarily observed in the city and districts, I have not taken into account some of less frequent occurrence, and which, from their prevailing almost everywhere—occasionally or frequently to as great an extent as they do here—cannot be regarded as depending on anything peculiar to our soil and climate. I allude more particularly to smallpox, measles, scarlatina, pertussis, influenza, cholera, erysipelas, &c., most of which prevail more or less frequently; assume, occasionally, an

epidemie and malignant character, and occasion a large increase in our ordinary mortality.

It is proper to remark, that the diseases mentioned do not prevail to an equal extent in all the parts included within the jurisdiction of the Board of Health. So far from it, they are mostly found to vary in relation to their origin, progress, and type, according to locality and surrounding circumstances. "In the compactly built portions of the city, disorders show themselves which are rarely seen in the surrounding suburbs, while the rural districts are visited with diseases which never originate in the heart of the city."¹ The city proper is entirely, and the compactly built portions of several districts are nearly clear of those forms of fever referable to a malarial poison. Most of the cases observed within its limits are traced to exposure elsewhere. The ordinary location of those fevers—remittents and intermittents, in all their grades of malignancy—is the thinly settled districts lying west of Broad Street, the low flat lands south of the city called the *Neck*, along the banks of the Schuylkill and Delaware, and various portions of the northern and northwestern districts. Exceptions to the exemption of the city from febrile complaints of malarial origin have recently been of rare occurrence—rare at least to such an extent as would amount to an epidemic. When such cases have occurred, the disease, differing in some respects from common remittents, has originated in and been limited to the confined and filthy alleys which abound in some city and district wards, and has prevailed among the lowest classes of the population; as was observed among the blacks in the summer and autumn of the year 1823, when they were visited by a severe and fatal epidemic.²

At an early period in the history of Philadelphia, the city plot, while subject to diseases arising from vicissitudes of temperature, was not, as now, exempt from malarial complaints. So far from it, the broken condition of the soil, the existence of numerous pools—natural or artificial—the decomposition of the timber cut down for objects of improvement, the exposure of a virgin soil to the action of the sun, the presence of creeks and rivulets with flat and marshy banks, and other natural and accidental sources of miasmatic exhalation, rendered it prone, unprotected as it was by the scattered nature of the buildings, to frequent, if not annual visitations of autumnal fever. Dr. Bond, writing as late as 1766, notices, as we have seen, the prevalence of these diseases along the Dock Creek, and the records of the city leave no doubt of the fact that the vicinity of the latter, and other marshy localities within the bounds of the city, were often the seat of the diseases in question. Dr. Rush has left us, in a distinct publication, the description of an epidemic of bilious remittent fever which prevailed in 1780. But even this fever may properly be ascribed, in great measure at least, to the miasmatic exhalations evolved, not within the limits of the then existing city, but in the vicinity, and carried to the former by the south and southwest winds which passed over the infected districts. Such, indeed, appears to have been the opinion of

¹ Tr. of Med. Soc. of the State of Penns., i. 29.

² See Emerson, Philad. Med. and Phys. Journal.

Dr. Rush, who, after remarking that these winds passed over the land lying between the city and the conflux of the two rivers, adds: "From the exposure of the district of Southwark (which is often distinguished by the name of the *Hill*) to the southwest winds, the fever made its appearance in that appendage of the city. Scarcely a family, and in many families scarcely a member of them, escaped it. From the Hill, it gradually travelled along the second street from the Delaware, improperly called Front Street. For a while it was confined to this street only, after it entered the city; and hence it was called by some people the *Front Street Fever*. It gradually spread through other parts of the city, but with very different degrees of violence. It prevailed but little in the Northern Liberties. It was scarcely known beyond Fourth Street from the Delaware."¹

To those who have paid attention to the subject, it can scarcely be necessary to remark, that in all our cities—the large ones particularly—remittent and intermittent fevers are limited to the suburbs. As the buildings extend out, and the closely inhabited portions expand, and by so doing lessen the area of humid and exposed soil, the disease recedes. Charleston, Savannah, New York, Buffalo, Auburn, Geneva, Syracuse, Salina, Louisville, and other places, may be appealed to in illustration. If the reader desires to have another convincing proof of the cessation of the production of autumnal fever from changes of the kind mentioned, let him come and see what has been the result in Philadelphia. In former days, when the city was of limited extent—with few improvements—with buildings scattered about, and leaving open and unimproved spaces between—with a marshy stream running through the greater part of it—with ponds, natural and artificial, spotted over the plot in various directions, and with unpaved streets—fever was of common occurrence, and epidemics were not unfrequent. At present, malarial fevers are unknown in the city proper, as well as in the compactly built and well-drained portions of the suburban districts. If we wish to meet with them, we must go to the outskirts of those districts, or to some distance beyond them, to the open meadow ground of the Neck, or to other unimproved surfaces of the vicinity; but more particularly to the marshes which still lie along the river banks. During the period embraced in the calculations of our townsman, Dr. Emerson, the fever, in some of its forms, was almost always present somewhere. But whilst in the more dense and well-paved parts the air seemed unusually healthy, remittents and intermittents, when they occurred there, could almost invariably be traced to night exposure in the country or suburbs. Great as was the amount of sickness during the epidemic of 1822–3, it was for the most part confined to the comparatively small proportion of population inhabiting the unpaved or ill-paved streets. Since the time to which this has reference, thirty years have elapsed. The compact, dense, and well-paved parts have extended far beyond where they had then reached; and with their expansion the disease has receded.

¹ An Account of the Bilious Remittent Fever as it appeared in Philadelphia in 1780, in *Med. Inq.*, ii. 232.

HISTORICAL SKETCH

OF THE EPIDEMICS OF 1699, 1741, 1747, AND 1762.

Having, thus far, disposed of various subjects intended as introductory to the main object of the present work, I now proceed, in pursuance of the plan proposed, to present an historical sketch of the several epidemics of yellow fever by which the city of Philadelphia has been visited. If, with what has been said on the subjects alluded to, we revert to the results of observation on the same points in tropical regions, and, indeed, in all localities where the yellow fever prevails frequently, or has prevailed occasionally, we shall have no difficulty in understanding that the disease may well have committed, and may continue, off and on, to commit a greater or less degree of mortality among us. Hence, the medical history of this country teaches us, that epidemics have been numerous, and that their origin would seem to have been contemporaneous with that of the city itself. That a difference in respect to the frequency of such visitations exists between the city in question and other parts further south, and especially tropical regions, is certainly true; for in the latter the disease is, as it were, endemic, the causes being permanent and operative at each revolution of the seasons, and giving rise, the assertion of Dr. Copland to the contrary notwithstanding, almost annually, on the arrival of susceptible subjects, to the fever; while, in some seasons, for reasons I need not mention here, it spreads widely and assumes the character of an epidemic. Here, on the contrary, the summers, though usually marked, while they last, by a degree of heat which is truly tropical, are comparatively of short duration, and never or seldom alike two successive years, a hot and dry, or a hot and wet summer being followed by a cool or wet summer, and each being, as we have seen, followed by a cold winter. From this, it follows that the fever is not, and cannot be endemic, and only appears at intervals of longer or shorter duration—disappearing for a few or even many years, and breaking out anew, either sporadically or in the garb of a wide-spreading epidemic.¹

Be this, however, as it may, the settlement of the city took place in or about the year 1682, and scarcely had a few hundred families from the mother country clustered together, and, clearing a small portion of the selected plot, provided themselves with comfortable dwellings, before an epidemic fever broke out among them, spreading desolation all around, and producing a mortality—to use the language of one of its delineators—fully equal to, or exceeding—if we take into consideration the then infant state and limited population of the place—any that has occurred at subsequent times. This earliest onset of the disease occurred in 1699, when Philadelphia, then but about seventeen years old, was little more in point of extent than an ordinary country town.

¹ See Med.-Chirurg. Rev., July, 1847, p. 207.

The disease, as I need scarcely remark, had long been known in the West Indies. In calling attention to this subject, I shall not enlarge on the fact that the early visitors and settlers of the colonies, and, indeed, the very companions of Columbus, suffered greatly from a fever which—judging from the imperfect accounts of it handed down to us by Herrera, Oviedo, Gomara, Peter Martyr, and others, and the circumstances connected with its origin and spread—bore a strong resemblance to the one under consideration. Still less will it be necessary to dwell, as some have done, on the events that occurred among the Indians of New England, anterior to the settlement of the Whites in 1620, when the tribes are said to have been reduced from 30,000 to 300, by a disease which bore in some respect a resemblance to the yellow fever;¹ for, though the sick are reported to have bled from the nose and turned yellow, like a garment of that colour, which they pointed out as an illustration, we cannot be censured for undue skepticism, if we entertain doubts as to its identity with true yellow fever, especially as it is acknowledged to have raged in winter. Laying aside all these equivocal instances of the early appearance of the yellow fever, we gather from the older travellers in, and histories of tropical regions, as also from the medical records of our own country, that the true disease had broken out, and spread extensively, prior to its appearance in this city, while it manifested itself simultaneously in some of the sister cities of the American colonies. According to Vines,² it appeared in Barbadoes in 1647. Ligon, who records the same fact, states that the inhabitants of the island, and shipping too, were so grievously visited with the plague (or as killing a disease), that, before a month had expired after his arrival, the living were hardly able to bury the dead.³ In speaking of the following year, 1648, Du Tertre says: “During this year, the plague, unknown in these islands since the time they were inhabited by the French, was introduced therein by some vessels. It commenced at St. Christopher, and, in the course of eighteen months, carried off one-third of the inhabitants.”⁴ Writing ten years later, Rochefort⁵ remarks: “The plague was formerly unknown there (the West Indies), as well as in China, and some other Eastern countries. But a few years ago, the greater number of these islands were afflicted with malignant fevers which the physicians regarded as contagious.” It prevailed in Jamaica in 1671.⁶ The event was connected with the return of the victorious fleet “from the signal Panama expedition,” when “they brought with them a high, if not pestilential fever, of which many died throughout the country.” The fever appeared twenty years later at Leogane (St. Domingo), in 1691, on which occasion, according to Moreau de St. Méry, it was brought by the fleet

¹ Hutchinson, i. pp. 34, 35, and Webster, i. p. 177, &c.

² Collection of Massachusetts Papers; see Bancroft, p. 316.

³ A True and Exact History of the Island of Barbadoes, &c., by Richard Ligon, pp. 21 and 25.

⁴ Hist. Générale des Antilles, i. p. 422.

⁵ Hist. Nat. et Morale des Iles Antilles de l'Amérique, 4to. p. 2.

⁶ Trapham, State of Health of Jamaica, 1679, p. 81.

of Admiral Ducasse.¹ The same year it showed itself at St. Christopher,² and the next at Port de Paix.³ The outbreak of the disorder, and its extensive ravages in Pernambuco (Brazil), from 1687 to 1694,⁴ is known to most readers; so, likewise, its supposed introduction thence into Martinique in 1690, by the *Oriflamme*, which there touched on her return from Siam.⁵ It prevailed also in the city of the Cape (St. Domingo) in 1696; and at Leogane in 1698.⁶ Hughes, in his *Natural History of Barbadoes*, writing on the authority of Dr. Gamble, mentions the prevalence of the fever in that island in 1691.⁷ It was then and there called the new distemper, or Kendal's fever, after a distinguished and popular officer of that name. Hughes also mentions its occurrence in 1696. According to Captain Philips, who visited the island in 1694, it prevailed then very extensively, and had done so during the war, some years before.⁸ "I shall," says the worthy captain, "pretend to give no account of this island, which is so well known, being one of our own plantations, but only observe that, though it be a pretty pleasant spot, and inhabited by a great many worthy hospitable gentlemen, yet it was its fate now to be violently infected with the plague, so that, in the late war, it proved a perfect grave to most that came there, all new comers being generally seized with the pestilence; of which very few recovered. Captain Thomas Sherman, in his majesty's ship *Tiger*, in the two years that he lay there, buried out of her 600 men, as he told me, though his complement was but 220, but still pressing men out of the merchant ships that came in, to recruit his number in the room of those that died daily. I lost about eighteen of my men by it, and, in truth, did not expect to escape myself, and therefore was so indifferent, that there was not a friend or acquaintance of mine seized with the distemper but I freely and frequently went to visit him, which probably was the reason that I escaped. Here died about twenty masters of ships during my stay here."

An epidemic of fever, upon the true yellow fever nature of which there can be little doubt, prevailed at Rocheford, in France, during the autumn of the same year, 1694.⁹ There is every reason to believe, indeed, that even in this country the fever had existed prior to its appearance in Philadelphia; for, in the summer of 1692, a sickness prevailed in Boston which, from the circumstances under which it spread, the period of the year at which it appeared, and the mortality to which it gave rise, we may conjecture to have been the disease under consideration. Our information on the subject is derived from Hutchinson, who, in his *History of Massachusetts Bay*,¹⁰ says: "In

¹ Hist. de St. Domingue, ii. p. 479.

² Ibid., vol. i. p. 701.

³ Ibid.

⁴ Ferreyra da Rosa, *Trattado da Constituçam de Pernambuco*. Lisboa, 1694.

⁵ Labat, *Nouveau Voyage en Amérique*, i. p. 72.

⁶ Moreau de St. Méry, i. p. 534; ii. p. 479.

⁷ Hughes's Hist. of Barbadoes, p. 37.

⁸ Philips, *Voyage to Africa and Barbadoes*; Churchill's Collection, vi. p. 257.

⁹ Chirac, *Traité des Fièvres Malignes; des Fièvres Pestilentiellles, et autres*, i. p. 30. Pringle, *Diseases of the Army*, p. 323.

¹⁰ Vol. ii. pp. 71, 72.

1692, it was resolved there should be an expedition the next year. A fleet was to be employed in the winter, in reducing Martinico, and, having performed that service, was to go to Boston, there to take on board a body of land forces, under Sir Wm. Phipps, and so proceed to Quebec. Had Quebec been the only object, and reasonable notice given to the colonies, there was reason to expect success. By the fleet's going to the West Indies, the whole design was blasted. If sickness had not weakened them, the force would still have been insufficient for the reduction of Martinico; but the mortality was so great, that before Sir Francis Wheeler, the commander-in-chief, came to Boston with the fleet, June 17th, he had buried 1,300 out of 2,100 sailors, and 1,800 out of 2,400 soldiers. It may be supposed the Admiral had done with the thoughts of the Canada expedition." Hutchinson adds, that the "distemper, which had been in the fleet, spread to Boston, and was more malignant than even the smallpox had been, or any other epidemical sickness which had been in the country before; and many families left the town and resided in the country until the infection ceased." In this instance, the cause of the disease was in all probability located in the vessels where it originated, and where the inhabitants of Boston, if they were really affected, received the infection. But the truth of this communication and spread of the disease is open to great doubt; for Cotton Mather, a contemporary of the event, says: "There was an English fleet of our good friends with a direful plague aboard, *intended* hither. Had they come, as they intended, what an horrible desolation had cut us off, let the desolate places, that some of you have seen in the colonies of the South, declare unto us! And that they did not come, was the signal hand of Heaven."¹ From this it would seem that Sir Francis Wheeler's vessels may have arrived at Boston most dreadfully infected, but that the disease was not propagated to individuals on shore.²

In the same year that the yellow fever made its appearance for the first time in Philadelphia, it prevailed to a considerable extent in Charleston, where, as we are told by Hewatt,³ it carried off a considerable number of people." "Never," says the historian, "had the colony been visited with such general distress and mortality. Few families escaped a share of the public calamity." Dr. Ramsay, in his *History of South Carolina*, alludes to this early epidemic, but is uncertain as to the precise year in which the disease broke out, mentioning that the event occurred in 1699 *or* 1700.⁴ But, independently of the statement of Hewatt, which seems to admit of no doubt, the matter is set at rest by a letter from Mr. Isaac Norris, of this city, who, under date of 18th September (9th month) old style, 1699, says to his friend, Jeffrey Pennell, that he had information from Charleston of the great mortality that had occurred there from the fever—one hundred and fifty having

¹ Magnalia, Book 7, p. 116.

² Webster, i. p. 208.

³ An Historical Account of the Rise and Progress of the Colonies of South Carolina and Georgia, i. p. 142.

⁴ Vol. ii. p. 82.

died in a few days, the survivors flying into the country,¹ and the town being thinned to a very few persons.

Of medical accounts of the epidemic of 1699 in this city, we are perfectly destitute; the references to, and descriptions of it which we possess—all of them meagre, and, in a professional point of view, void of interest or of usefulness—being gleaned from the journals of the few travellers who visited our shores, the manuscript correspondence of one or two citizens of the place, and the pages of the early historians of the colonies.²

Mr. Isaac Norris, already mentioned, an eye-witness of the event under consideration, in a letter to Jonathan Dickinson—for a perusal of which I am indebted to his distinguished great-grandson, Dr. G. W. Norris, of this city—states, under date of 15th of June: “Our families, among which I include thy own, are well by the divine mercy of the Lord, only my poor wife begins to complain this evening, which is some trouble, because some are sickly; and lest the news (as such generally does) should magnify ere it reach you, this my advice, that it has been pretty sickly this month, many children have died, and this last week many older persons have been taken ill.” “I think it is exactly the Barbadoes distemper; they vomit and void blood.” Writing to the same on the 24th of the month, Mr. Norris mentions the death of many of his acquaintances and friends, and adds: “But we hope (the weather now being pretty cool and pleasant) that it may be a means, in God’s hands, to stay the distemper; and though some have been thus visited more than ordinary, yet, in the general, it is not so sickly as is represented abroad.” “This has been, about harvest time, the hottest summer that I ever felt; I really think several died in the field with the violence of the heat.”

Another letter writer, William Monington,³ confirming the last, says:—“Our arrival here was in the beginning of corn harvest; the weather grew very hot and fainty; and some of our passengers that went presently to harvest work, spent themselves in the heat, and expired in the field.” The disease, as we glean from the letters of Mr. Norris, broke out about the beginning of June. On the 1st of July, the same worthy citizen writes to Mr. Dickinson: “The sickness did once seem to abate, but is now more raging and mortal. I shall not say much, knowing such things fly fast enough, and is seldom lessened but magnified, but submit to the will of our almighty and wise God, who is now showing his power, in order, I believe, to our humiliation.” On the 8th of July he again writes: “Trade looks very dull, occasioned chiefly by the extraordinary visitation it has pleased the Almighty to afflict this poor place with. I never knew the like since I came into the country; it has been sometimes sickly, yet I do not remember it ever so mortal. Many have died, Friends and others, and many now very ill.” “Some were in hopes,” he says, in a letter to John Mills, 9th of 8th month

¹ Webster, i. p. 214. Rush, Epid. of 1793, p. 153; and Med. Inquiries, iii. p. 108.

² Webster, Hist. of Epidemics, i. p. 211; Rush, Epid. of 1793, edit. of 1794, pp. 134, 182. See also Proud., Hist. of Penn., i. 431; Pemberton’s Letter to Dr. Wistar; Additional Facts and Obs. by College of Phys., p. 5.

³ William Monington’s Letter to Andrew Russell, Med. Mus., i. p. 227.

(August), "that the cool weather might, by Divine permission, help us, but cannot say the distemper much abates; five or six were buried on seventh day, and three on first day." But, on the 22d of 9th month (September), he writes to John Askew: "I now think thou wilt be glad to hear it has pleased God to withdraw his hand, and put at present a stop to our sore visitation, and the town and country generally healthy. I thought, but have not time, to have sent thee a list from the registry of the dead. The number is about 220 in this town, 80 or 90 of them Friends." And on the 5th, 10th month, in writing to Jonathan Dickinson, he says: "Our place, through great mercy, very healthy again, and an extraordinary moderate and open fall."

Thomas Story, who, in company with his friend, Roger Gill, travelled in this country during the course of that year, and afterwards, settling in Philadelphia, became one of the provincial council, and subsequently Recorder of the city, has left us, in the journal of his life, a gloomy picture of the distress occasioned by the distemper, and of the extent of its ravages. On their way to the city from the eastward, they heard of the sickliness of the place; but, undeterred by the melancholy tidings they received, they entered the infected town, where they heard more of the disease, many Friends being *on their sick and dying pillows*. Such, he says, was the infection, that some Friends wrote to Burlington, in an answer to a proposal to adjourn the meeting of Quakers about to take place in the city, that though it was impossible to do so till the meeting had come together, yet "it might do well to discourage, in all places, the greater number of young people and servants that usually come to town on those occasions, because of the great infection, and incapacity of friends and inns in town, at such a juncture, to lodge and entertain them, there being few, if any, houses free of sickness" (p. 223-4). Story and Gill remained some time in the city, "visiting and encouraging the sick and afflicted." The latter was taken sick with the common distemper, and died on the seventh day.¹

The city, at that time, was necessarily of very limited extent. Houses were scattered in various directions about the plot which, as we have seen, had been early surveyed; but the principal and more compact improvements did not reach much beyond the vicinity of the Dock, where the settlement originally commenced, and along the margin of the river, at several points of which dwellings and stores were erected. The ground had been but recently cleared; the creeks, pools, and swamps were much as nature had made them, and exposed to the drying action of the scorching sun. The number of families already gathered together within the limits of the infant city, did not much exceed, according to the estimate of some writers,² six hundred. Supposing each of these families to have consisted of 6.35 persons, we have a total population of considerably less than 4,000 inhabitants.

¹ A Journal of the Life of Thomas Story, containing an account of his remarkable convincement, and embracing the truth as held by the people called Quakers: Newcastle upon Tyne, p. 223, fol. 1747. Gough, History of Quakers, iii. 516.

² Currie, Feb. of 1799, p. 38.

The epidemic of 1699 was preceded by one of influenza.¹ Like those that have occurred subsequently, it limited its ravages almost exclusively within the precincts of the infant city, and extended but little, if at all, into the country.² But within those circumscribed limits, and among this scattered and small population, its effects appear to have been unusually severe. Story states that the mortality amounted to six, seven, and eight a day for several weeks, and that ten were buried in one day.³ "From the beginning of the sixth month," says Monington, "to the latter end of the eighth month, there died of Friends and others (besides those that were buried in the churchyard) to the number of 170, men, women, and children; but afterwards the violence of the distemper abated, and very few died." According to Mr. Norris, forty died from the 15th to 27th of July (Letter to James Mills). On the next day he says to Thomas Lloyd: "Death still knocks at our doors, and there is not a night or day (I think) but several die. As I hear, the grave-digger's account is 150." And, as we have seen, he subsequently stated that the whole number of deaths amounted to 220, of whom 80 or 90 were Quakers. Assuming this as the correct number, and estimating the population at 3,800, we shall find that the fever carried off 1 out of every 17.3 of the inhabitants; a mortality little, if at all, inferior to that of subsequent epidemics. Of the proportion of deaths to the number attacked with the disease, we learn nothing very definite. It is presumable, however, that it was very large, as M. Monington states that "few ever lived seven days after they were taken ill, and very few recovered." Mr. Norris, in like manner, limits the duration of the disease to seven or eight days, adding: "I think the tenth person hardly escapes" (Letter of 8th July), and that some were "well, and dead in four days" (31st June).

Well can we understand the dismay which this wide-spreading mortality created among the peaceful inhabitants of the rising city, as strongly depicted by Story. "Great," he says, "was the majesty and hand of the Lord; great was the fear that fell upon all flesh; I saw no lofty, airy countenance, nor heard any vain jesting to move men to laughter; nor witty repartee to raise mirth; nor extravagant feasting to excite the lusts and desires of the flesh above measure; but every face gathered paleness, and many hearts were humbled and countenances fallen and sunk, as such that waited every moment to be summoned to the bar, and numbered to the grave."

The authorities we have in relation to this epidemic being of the unprofessional character mentioned, we are left much in the dark relative to the nature and phenomena of the disease. But, imperfect as the accounts we possess are, enough may be gathered from them, aided by concomitant circumstances, to enable us to arrive at a tolerably satisfactory conclusion on the subject. Mr. Norris, who, as seen, describes the fever as the Barbadoes distemper, says that the patients vomited and voided blood, that they were generally taken with a shivering, which was quickly succeeded by a violent fever, which, after the first day, seemed to abate, yet left a violent pain at the sto-

¹ Webster, i. p. 212.² Story, 223.³ Ibid.

mach, and sometimes in the head, back, and bones (8th July); but that they were generally sensible to the last (27th July). While Monington, entering also into some details, states that "it seized with a violent pain in the head and back, and caused vomiting of blood;" and that death took place within the seventh day after the attack. When, with these symptoms in view, we bear in mind the period of the year at which the disease broke out, the effect of cold weather in arresting its progress, the nature and condition of the localities visited, the extent of the mortality, the excessive heat of the summer, the inference is natural, that the fever differed in nothing from that which, under the name of yellow fever, has subsequently prevailed in the same place. The chroniclers of the epidemic say nothing of its causes, and give no intimation of its being communicated from countries abroad by contagion. Nowhere, so far as I have been able to ascertain, do we find a suggestion that any vessels had arrived from the West Indies with the disease on board, or in a condition calculated to infect the city, unless we place in that category the *Britannia*, which reached the city on the 24th of June after a long passage, with two hundred individuals on board, having lost fifty by death, and containing many sick. But the *Britannia* came from Liverpool, and the disease from which it suffered could not have been the yellow fever, and was, in all probability, ship typhus; or, as it was then frequently called, the palatine fever. But Gough,¹ and Proud,² who came long after them, calling attention to the fact that the disease had either long before prevailed, or continued to prevail, in the West India Islands, threw out the idea that it did not arise from local causes in Philadelphia, but was the offspring of foreign importation; while Pemberton, more explicit than the rest, states positively "on information derived from his father, that it was imported in a ship (or other sea vessel) from the island of Barbadoes, whose cargo consisted of cotton in bags, which were landed at a wharf between Market Street and the drawbridge, and there stored for sale."³ Perhaps the correctness of the inference will not acquire probability when the reader is told that Pemberton's father, from whom the facts are derived, was only fifteen years of age at the time of the calamity. But we are assured that young though he might be, "he was capable of making his remarks on occurrences, and afterwards of remembering them;" and that he "knew it to be the invariable judgment of the physicians and other citizens, that the disease was introduced among them in the manner related above, attributing it to no other visible cause."⁴

¹ *Op. cit.*, iii. 516.

² *Op. cit.*, i. 431.

³ Additional facts by the College of Phys., p. 5.

⁴ Dr. Rush, in his account of a subsequent visitation, and Mr. Noah Webster, in the first volume of his well-known work on epidemics, frequently refer to the correspondence of Mr. Norris—from which, indeed, they derive the principal share of their information respecting the events of 1699. They both state having had access to the MS., and yet they both make the most remarkable—and, what is very extraordinary, in some instances the same—blunders as to the dates of the letters. These blunders are not of a trifling and unimportant character, leading, as they do, into a false conclusion as to the period at which the epidemic broke out, and terminated. Thus, Mr. Webster represents Mr.

From 1699 to the year 1741, we hear nothing of the yellow fever in Philadelphia. The records of the period show that it prevailed, with more or less severity, at Martinique in 1703 and 1706;¹ at Cape François, now Cape Haytien, in 1705, 1723, 1733, 1734, 1739, and 1740;² at Leogane in 1732;³ at Carthagená in 1729 and 1730;⁴ at Barbadoes in 1723 and 1733.⁵ At Charleston it prevailed in 1703, at which time the inhabitants were apprehensive of an invasion from the French and Spaniards.⁶ It broke out again with great violence in 1728, when, according to Hewatt, it swept off multitudes of the inhabitants, both white and black, the physicians not knowing how to treat the uncommon disorder, which was so suddenly caught and proved so generally fatal, and few being able to give assistance to their distressed neighbours, however much needed and earnestly desired.⁷ It again appeared in 1732, 1739, and 1740.⁸ Nay, the disease appears to have shown itself nearer to this city; for, in a letter written by the late venerable Dr. John Bard, of New York, we are told that he had heard, from the ancient inhabitants of that city, that, so long ago as the year 1702, a malignant fever, little inferior to a plague, was imported into it, which, from its extreme mortality, was distinguished by the name of "the great sickness."⁹ Dr. Bard adds:

Norris as writing on the 15th of *August* that the fever had broken out about the beginning of that month; Dr. Rush mentions the 12th of August, O. S. We have seen that the announcement was made on the 15th of *June*. The letter stating that the distemper appeared to abate at one time, but afterwards revived, is wrongfully dated by Webster 1st *September*. It was written on the 1st of *July*. Instead of announcing, on the 1st of *October*, as stated by this writer, that some had hoped the cool weather would have relieved the city, Mr. Norris did so on the 9th of *August*. The information as to the cessation of the epidemic was conveyed under date of 22d September, and not of 22d October, as stated by Webster, and 9th of November, O. S., by Dr. Rush. Mr. Norris refers to the intense heat of the time, not on the 24th of August, as Dr. Rush says, nor on the 1st of September, as might be inferred from the words of Webster, but on the 24th of June. Finally, the existence of the fever in Charleston is mentioned in a letter dated 18th September, and not 18th October, as Webster has it, or 18th of November, as stated by Dr. Rush.

The dates, as given in the text, were carefully noted from a correct copy of the letters in the handwriting of the late Mr. Isaac Norris. They harmonize with well-known facts, and correspond with the contents of Mr. Monington's letter. Roger Gill returned to the city after the disease had already prevailed some time and spread widely. He was stricken down by it, and died on the 3d of August, after an illness of seven days. The epidemic, therefore, could not have broken out in the beginning of that month; it must have done so long before. On the other hand, Monington, as we have seen, states that the mortality from the beginning of 6th month (June) to the end of 8th month (August), was 120.

¹ Feuilleé, Journal d'Obs. dans la N. Espagne, &c., pp. 186, 187.

² Moreau de St. Mery, i. pp. 534, 535; Desportes, Malad. de St. Domingue, i. p. 191.

³ Moreau de St. Mery, ii p. 479.

⁴ Ulloa, i. pp. 41, 44.

⁵ Warren, p. 4.

⁶ Hewatt's Hist. Acct., i. 182.

⁷ Ibid., p. 317; Lining, Edinb. Essays, &c., ii. p. 407.

⁸ Lining, Ibid.; Ramsay's Hist., ii. pp. 83, 84; Simons, Carolina Journ., i. p. 2; Ibid., Report to Board of Health, p. 6.

⁹ Carey's American Museum for 1788, p. 454; Currie, Dis. of the U. S., p. 63.

“This formidable disease, if tradition says true, was brought here (New York) from St. Thomas in a single bale of cotton.”

Though enjoying as free an intercourse as before with the West Indies, and the other North American colonies, during the long period above mentioned, Philadelphia remained unseathed until 1741. But in the latter year, while prevailing in Jamaica and other West India islands,¹ the disease broke out, as it would appear, with violence, and continued until checked by the advent of cold weather. Our information relative to this epidemic is, perhaps, even more unsatisfactory, in a professional point of view, than that we possess concerning the former. Of the localities it affected; of the peculiarities of the season; of the condition of the city at the time of its prevalence; of the precise ratio of the mortality it occasioned, our information is very limited. But that the disease did really prevail, that it occasioned no inconsiderable loss of life among the inhabitants, and presented many, at least, of the usual characteristic phenomena of the yellow fever, cannot be a matter of doubt. I am perfectly aware that Mr. Pemberton, a respectable citizen of this city, whose letter to Dr. Wistar has already been cited in reference to the epidemic of 1699, distinctly ascribes (p. 6) the fever to the year 1740 instead of 1741. But, as he nowhere refers to the disease of the last-mentioned year—which, nevertheless, is spoken of by medical and other writers of the times and public documents—and as he is sustained by no other authority in affixing the above date to the disease, we may reasonably presume that his memory did not serve him right on the occasion, that the year 1740 was free from an epidemical visitation of the fever, and that his remarks have reference to the disease of the succeeding year.

Watson, in his *Annals of Philadelphia*, gives us some useful information on the subject, which he derived from letters addressed at the time by Mr. Peters to the proprietary, then in England. These letters, while fixing the date of the visitation, tell us of the havoc it committed, the name by which it was known, the class of individuals on whom it more particularly bore, as well as the panic it occasioned. “It was called the ‘palatine disease, because prevailing among the German emigrants, probably from their confinement on shipboard. The inhabitants were much alarmed, and fled to the country towns and places; and the country people, in equal fear, avoided to visit the city” (ii. p. 371).

For the following letter of Mr. Peters, I am indebted to my friend Dr. Norris. It is dated Philadelphia, Oct. 20, 1741, and is as follows: “At the time of your brother’s departure from this city, it was afflicted with a most malignant distemper imported in the spring by ships from Ireland. At first, it crept along the wharves, affecting strangers, common sailors, and the low and poor part of the people, and was easily managed by the physicians; but, as the dog-days advanced, having a long and dry season without gusts, the sickness increased and attacked the better sort of the inhabitants, of whom a good many died, though not in such numbers as is reported. About the 17th

¹ Hume, p. 230; Williams, p. 11.

August, the physicians opened the body of a man who died in forty-eight hours from the first sensible attack, and found his bowels and stomach quite mortified. From the 17th August to 17th September, the distemper continued very violent, and baffled all the arts of the docters. I stayed a few days at New York after your brother went, and there, by accident, I lighted on Dr. Warren's treatise of the yellow fever at Barbadoes, a book well known in London, and finding his description answer in most points to the Philadelphia sickness, I dispatched it immediately to Dr. Grœme, who was much assisted by it; and, after the arrival of the book, the distemper came to be well understood, so as to yield soon, in very violent cases, to a free use of sudorifics—Dr. Warren's specific for the cure. One partiular symptom attended the fever in Philadelphia which I suppose never appeared in Barbadoes, and that is a suppression of urine from the first attack. This was the case of poor ——; and when the urine was suppressed, there is no instance of any one's recovery. At present, we enjoy good health, and a fine season. According to the best accounts I am able to get, the number of the dead since the 1st of June amounts to no more than 250 "

Dr. Thomas Bond, a distinguished practitioner of that period, and whose name, conjointly with that of his brother, Dr. Phineas Bond, is found often honourably associated with that of our illustrious Franklin in works of benevolence and public utility, in the introductory lecture to a course of clinical medicine, delivered in December, 1766,¹ mentions his having witnessed the yellow fever five times since settling in Philadelphia, and adds: "It was in the year '41 I first saw that horrid disease." From Dr. Rush² we learn that the fever in question was referred to in a letter (now no longer to be found) of Dr. Franklin's to his brother, who stopped at Burlington, on his way from Boston to this city, until assured by the doctor that a thunderstorm, which had cooled the air, had rendered it safe to come into the city. It is also referred to in a memorandum appended by Dr. Kearsley, Sr., to the MS. copy of Dr. Mitchell's account of the epidemic of Virginia in 1741, and which, after being quoted by Dr. Rush (p. 134), was published in Coxe's *Medical Museum* (vol. i. pp. 20, 21), and subsequently in the *Medical and Philosophical Register* (vol. iv. p. 246, note). Dr. K. was long a practitioner in this city; and speaks of the disease as an eye-witness. But the most positive proof of the prevalence of an epidemic, and, at the same time, of its ravages, is found in the register of the Quaker's cemetery. Under date of sixth month (O. S.), Rush says, it is there stated that "a malignant fever now spreads much;" and, a month after, "many who died of the above distemper were persons lively and strong, and in the prime of their time" (p. 134).

The period of the year at which the fever made its appearance, the extent of the mortality it occasioned in a few short months, the effect of cold in arresting its career of destruction, the analogy of the symptoms to those described by Dr. Warren, of Barbadoes, the occurrence and fatal tendency of suppression

¹ Published from the Minutes of the Pennsylvania Hospital, in N. A. iv.

² Fever of 1793, p. 134.

of urine, the locality where it broke out, the manner in which it spread, the rapidity with which some of the cases ended fatally; all these circumstances—as well as the fact that while it raged here a disease of kindred nature prevailed, not only in the West Indies but on this very continent, and at no great distance from this city (in New York¹ and Virginia²) which so little resembled the ordinary fever of the season as to take the physicians by surprise—lead to the conclusion that the disease was, notwithstanding the name of “palatine” given to it by some, analogous to that which had prevailed in 1699, and, like it, was entitled to the name of yellow fever. But we possess stronger proof to that effect; for, in the memorandum already mentioned, Dr. Kearsley has left us a sufficiently copious account of the symptoms of the disease to enable us to form an idea of the class to which it belongs. The paucity of materials we possess on the subject must be my apology for inserting this memorandum, notwithstanding the rather antiquated pathological views it exhibits. Comparing the disease with that described by Dr. Mitchell, of Virginia, Dr. Kearsley says:—

“1. Wandering pains, like those attending a rheumatic fever, but much more severe, were generally much complained of from the first by those who had the disease in Pennsylvania. They are not mentioned by Dr. M.

“2. A very great anxiety, with sickness and pain in the stomach, attended with an excessive convulsive vomiting, which no medicine would scarce relieve. This appeared on the first and second day, but more commonly on the third, when it was generally fatal, by bringing on hiccough, inflammation of the stomach and viscera, with a large discharge, by vomit, of a black, atra-bilious matter, like coffee-grounds, mixed with bloody lymph or coagulated blood; which frequently put a period to the patient’s life, though some recovered under this symptom by an early discharge of the black matter by stool.

“3. The atra-bilious humour, as Dr. Mitchell calls it, was highly acrid, yet not so viscid as that in Virginia, which gave it a more easy passage through the biliary ducts; and, being thereby more easily pumped up by the convulsive retchings of the stomach, hence, by its greater acrimony, it rendered this symptom more violent and fatal than it seems to have been in Virginia.”³

No one at the time seemed, as it would appear, inclined to look for the cause of the epidemic in the local sources of infection existing in the city. It was found more natural to attribute its development to importation from abroad. But, as generally happens in such cases, those who, not being able to discover in and about the city circumstances calculated to produce the fever, referred it to a foreign origin, differed as to the place whence it was to be traced, and the mode of its introduction. Mr. Pemberton, recalling the fact of a military expedition which proved unsuccessful, “being promoted a short time before by Great Britain against some of the West India possessions, particularly Carthagena,” adds that “troops were raised in Pennsylvania, and

¹ Colden, New York Med. Repos., xiv. pp. 1, 156. ² Mitchell, Med. Mus., i. p. 1.

³ Loc. cit., pp. 20, 21.

a communication, before utterly restrained, was opened with our city; and on the return of the ships and people employed in that business, a new species of fever was introduced." Dr. Lind, on the other hand, relying on information derived "from a gentleman in Philadelphia who had been a sufferer by the disease," states that the yellow fever was introduced "by means of a trunk of wearing apparel belonging to a gentleman who had died of the fever in Barbadoes;" whilst Dr. Bond, with much confidence, discovered the cause of it in a number of convicts from the Dublin jail; and others, again, as we have seen, traced it to German emigrants recently arrived from the palatinate. We shall see, in a subsequent chapter, that while such was the state of professional and general opinion on the subject in this city, a somewhat different view was upheld in reference to the origin of the same disease that year in New York and other places.

We next hear of the appearance of the fever, in an epidemical form, in 1744, when it prevailed likewise to a considerable extent in the city of New York.¹ It may be proper to state, however, that the existence of the disease at the period in question is open to some doubt. The only authority on the subject I have been so far able to discover, is Mr. Pemberton, who, in his letter to Dr. Wistar, already several times quoted, after stating that the fever last mentioned occasioned little alarm, continues: "Nor was the city, in that manner, remarkably affected until the year 1744, when a disease appeared with unusual symptoms, and proved mortal to a considerable number of its inhabitants, of whom divers were respectable characters, and was distinguished by the name of yellow fever, and known to be imported from some of the West India Islands." I may remind the reader that Dr. Bond, writing in 1766, mentioned his having witnessed five visitations of yellow fever, the first being in 1741. Such being the case, the statement of Pemberton may be thought to be in some measure sustained; for, from circumstances to which attention will be called presently, from the number of epidemics which occurred from 1741 to the period at which Dr. Bond wrote, and from his remarks relative to the subsequent prevalences of the disease, and to the courses and progress of the one in question, it is not beyond the range of possibility that the second epidemic he witnessed occurred in 1744. Nevertheless, however well founded this supposition may be, the statement of Pemberton is strongly counterbalanced by the direct testimony of Dr. Franklin, who closed a letter to his father, under date of September 6, 1744, with these emphatic words: "We have had a very healthy summer, and a fair harvest." Unless an error has crept into the date of this letter—an occurrence not unknown in Sparks's edition, from the seventh volume (p. 16) of which the above is quoted—or, unless the disease, if it occurred, did so later in the season than usual, Dr. Franklin's words set the matter at rest, as it is scarcely possible that one so alive, as he evidently was, to public concerns and events could have been so explicit as to the healthfulness of the season in

¹ See Bard's letter to Dr. Hosack, Add. Facts, p. 17.

question had the yellow fever prevailed to a sufficient extent to assume the character of an imported and wide-spreading epidemic.

Very different is the character of the testimony we possess relative to the prevalence of the yellow fever a few years after the last-mentioned date—in 1747; for although on this, as on former occasions, we must be contented with the statements of a few unprofessional writers, yet these statements leave no doubt as to the epidemic development of the disease in that year; while they furnish us with some insight into the circumstances under which it prevailed, and the causes to which it was generally ascribed. The testimony on which we rely consists of a letter from Dr. Franklin¹ to his mother, under date of October 16; of Mr. Pemberton's communication to Dr. Wistar, already used in reference to former epidemics; and of letters of Lynford Lardner² to correspondents in England; of Thomas Willing³ to the late Dr. Griffiths, of this city; and of Benjamin Chew to Dr. Wistar.⁴ The disease is also noticed in some letters of the distinguished American botanist, John Bartram, which were placed by his family in the hands of the late Professor Benjamin Barton, and by the latter inserted in his *Philadelphia Medical and Physical Journal*.⁵ It is likewise referred to by Mr. David Palmer, who, in a letter to the late Mr. Ed. Penington, of this city, dated 6th month (June) 29, 1747, says: "People are exceedingly sickly in many parts of the country, and especially in this city, where upwards of twenty are frequently buried in a day; the yellow fever being very brief, carrying off healthy persons in two or three days. I heartily wish we would both escape the dangers with which we are threatened, that we may once more meet," &c.⁶

As on other occasions, the fever, in 1747, broke out during the hottest part of summer. As just stated, it already existed at the close of June. Lynford Lardner, writing under date of 3d September (O. S.), informs his correspondent that the fever had already then been raging for some time. How long it continued to do so we are not exactly informed. All we know is, that on the 24th of September it was still prevailing in town; and we may infer, not only from the course the disease has pursued at other times, but from Dr. Franklin's letter, that it continued to prevail till arrested by the accession of frost. In this letter, which, as stated above, bears date of 16th October, the doctor says: "Besides the measles and flux, which have carried off many children, we have lost some grown persons by what we

¹ Mr. Sparks, in his beautiful and valuable edition of Franklin's works, vol. vii. p. 41, has ascribed this letter to the year 1749; but I have reason to think it was written in 1747. 1st. In Duane's edition (vol. vi. p. 8) of those works, it is placed under the date of the last-mentioned year. 2d. The yellow fever did not make its appearance in Philadelphia in 1749; whereas we know it did so two years before.

² See Currie on Yellow Fever of 1793, where this letter is given in full, p. 5; Barton's Journal, i. p. 136.

³ Additional Facts, pp. 8, 9.

⁴ Ibid., pp. 11, 12; Webster, i. 240, alludes to the epid. of 1747.

⁵ Barton, Philad. Med. and Phys. Journal, ii. part 2, p. 89.

⁶ Ibid., ii. p. 89.

call the yellow fever; though that is almost, if not quite over, thanks to God."

The disease was mostly confined within the limits of the southern part of the city, below the drawbridge, and at lodgings for sailors, and in the neighbourhood of the dock, which was then uncovered.¹ As to the mortality it occasioned, we know but little; but there is reason to believe that, though, as Mr. Palmer stated, it frequently occurred that twenty were buried in a day, it was not great—much inferior, both numerically and in comparison to population, to what it had been in some of the former, and has been in subsequent epidemics. Mr. Willing says that it caused the death of several men of standing, and about forty others of those in the neighbourhood of the bridge; but, as the fever extended its ravages below the dock, we may presume the number of deaths from it was larger than here mentioned. Be this as it may, the fever became soon the source of great alarm among the citizens, and engrossed the conversation in all companies. So early as the 3d of September, Philadelphia, according to Lardner, had become a melancholy place, and many, whose business and family would permit them, fled the city, and sought refuge in the country.

The last-mentioned writer is the only one who has left the least intimation of the symptoms by which the fever was characterized, and even his statement on the subject is imperfect and unsatisfactory. Were it not, indeed, assisted by the concomitant circumstances already mentioned, it would scarcely be sufficient to enable us to recognize the true nature of the disease—all he says being, that the latter admitted of no relief, cure, or abatement, never intermitted to the last moment of life, and specifying among the symptoms a pain in the head, and black vomiting, &c.

As to the causes of the epidemic, its origin, and mode of propagation, the public mind, judging from the letter-writers referred to, seemed to have been more fully made up than it had been heretofore. The disease is said to have exhibited contagious properties, and to have been imported from abroad. Pemberton contents himself with giving it, in general terms, a West India origin. Others were more explicit as to the particular mode of its introduction. Mr. Chew, in a passage I insert, not as expressing his individual sentiment on the subject, but that of the public generally and of the medical practitioners of the day, says: "I well remember it was the universal opinion that the disease was imported in a vessel from some part of the West Indies, and was communicated by the clothing contained in the chest of a person who died of the disease in the West Indies (Willing says Barbadoes); and that the person or persons who were present at the opening of the said chest were the first who were taken with the yellow fever; and to some or all of them it had proved fatal. I also perfectly remember that Drs. Th. Bond and Phineas Bond, Dr. Graham, and Dr. Cadwalader, with whom I was particularly acquainted, were all of them of opinion that it was imported in the manner above stated; and, until the year 1793, I do not recollect to have ever heard

¹ Pemberton, pp. 6, 7; Th. Willing, p. 9.

it doubted, whether the yellow fever, at the different times it had heretofore made its appearance in this city, was imported or had originated here" (p. 12).

I presume this to be the third epidemic which Dr. Bond says he saw. But, if so, he does not explain the introduction of the disease in the city in the same way as Mr. Chew and others, for he says: "It was generated on board of crowded ships in the port, which brought in their passengers in health, but soon after became very sickly." "I here," continues Dr. Bond, "saw the appearance of contagion like a dim spark, which gradually increased to a blaze, and soon after burst into a terrible flame, carrying devastation with it, and, after continuing two months, was extinguished by the profuse sweat of tertian fevers. But this is not the ordinary course of the contagion; it is usually checked by the cool evenings in September, and dies on the appearance of an October frost."¹ No mention is made by any of the writers who refer to this epidemic, of the particular vessel from which the disease could have been derived. It is true, that in the month of September the ship *Eurayle*, Capt. Cox, arrived from Barbadoes, where the fever was said to prevail, and that in consequence of this and of the fact that during the passage some of the crew had died of an acute disease, which was believed to have been the yellow fever, suspicion arose as to the vessel being the means of conveyance of contagion, and she was ordered down below the city and placed in quarantine. But the suspicion was a groundless one, for the fever had commenced to prevail long before the arrival of that vessel.

After an interval of more than ten years, the fever broke out in 1760, but in such a way as scarcely to merit the name of an epidemic, for the only writer who takes notice of it adds that it was not of long duration, nor extensive in its progress.²

More satisfactory is our information respecting the occurrences of 1762, when, according to the same authority, the same kind of disease again appeared, with a greater degree of malignity, attended with such symptoms as proved superior to the skill of the most experienced physicians.³ The disease made its appearance at the usual period of the year; was preceded, as all epidemics of the kind usually are, by very hot weather; was circumscribed within such localities as it has ever been found to prefer; and was arrested by the accession of winter. Dr. Redman, a distinguished practitioner of the time, has left us, in a letter addressed to the College of Physicians in 1793,⁴ an account of its origin and progress. He states that he had one patient as early as the 20th of August, whose case he treated at first as a remittent fever, from the circumstance of the individual living above a mile from the city, to the southward, near low and wet ground, though he sometimes came to town, and transacted business near the place where the yellow fever had then just begun to appear. Dr. Redman had not then seen any of the cases. "I had another patient on the 28th of August; from thence to

¹ *Introd. Lect. N. A. Journal*, iv. p. 271.

² *Pemberton*, p. 7.

³ *Additional Facts*, by the College, p. 7; see, also, Mr. Willing's Letter, *ibid.*, p. 10.

⁴ *Additional Facts*, &c., p. 19.

September 1, four or five. From that time, they daily increased; so that, from the 20th to the 25th of September (when the disease appears to have been at its height), I attended daily eighteen or twenty patients, though other physicians had more; and I believe Dr. Bond and others had patients in it about a week before me. From the 27th, the disease appears to have declined, so that, by the 20th of October, I had but two or three fresh patients in it, and then, I believe, only such persons as had lately come to lodge or live in that part of the town. After this, I had a few more scattered patients, and also two in the first week of November, but none afterwards." (p. 20.)

The disease was mostly circumscribed between Pine Street northerly and three or four squares southerly, and extended from Front or Water to Third or Fourth, in a westward direction. It made its first appearance, and committed the greatest ravages, about what is called the New Market (situated in Second Street, and extending southwardly as far as Cedar), and the square to the eastward of it. Within that space, after "considerable search and tracing, it was found to have originated in a number of small back tenements, forming a kind of court, the entrance to which was by two narrow alleys from Front and Pine Streets, and where sailors often had their lodgings."¹ Mr. Thomas Willing, in the letter already cited several times, speaks of the disease having prevailed extensively, and with considerable mortality, on or about the "sugar-house wharf," situated below Cedar Street.

We have no precise information as to the mortality resulting from the disease on the occasion before us. Mr. Willing estimates the loss in the vicinity of the wharf just named at upwards of sixty; but this estimate must fall far short of the number of those who fell victims to the disease, for the late Dr. Rush, who, in 1762, was studying medicine with Dr. Redman, states—in a note-book kept at the time, and of which he gives an extract in his account of a subsequent epidemic—that the disease spread like a plague, carrying off daily, for some time, upwards of twenty persons. When, with this in view, we bear in mind that the number of cases in daily attendance must, judging from the expressions of Dr. Rush, and the statement of Dr. Redman, have been considerable; that the disease proved highly malignant, and prevailed during more than three months, we may naturally infer that the mortality was very large—amounting, probably, to several hundred.

It would be in vain, at the present day, to look for a full and complete account of the phenomena presented by the disease during the epidemical visitation in question. Few, if any, of the practitioners of the time appear to have noted down the results of their clinical observations; or, if they did, their records have not reached us. All we possess on the subject is contained in a short extract from the above-mentioned note-book kept by the late Dr. Rush; and, also, in the short sketch of that occurrence communicated to the College of Physicians by Dr. Redman, to which reference has already been made. From the former, we learn that the patients were generally seized

¹ Redman, *op. cit.*, p. 21.

with rigors, which were succeeded by a violent fever, and pains in the head and back. The pulse was full, and sometimes irregular. The eyes were inflamed, and had a yellowish cast, and a vomiting almost always attended. The third, fifth, and seventh days were mostly critical, and the disease generally terminated on one of them, in life or death. An eruption, on the third or seventh day, over the body proved salutary. An excessive heat and burning about the region of the liver, with cold extremities, portended death to be at hand. (*Epid. of 1793*, p. 14, ed. of 1794.) That it would be difficult to recognize in this short and imperfect description a picture of the true yellow fever of subsequent years, will be at once admitted by all who are familiar with the disease; yet when—bearing in mind that Dr. Rush had not yet completed his medical studies, and may very naturally be presumed to have omitted some important and characteristic symptoms—we take into consideration the concomitant circumstances already adduced in reference to former epidemics, we shall find no reason for doubting the analogy of the disease with the true yellow fever.

Dr. Redman's description, though short, is more satisfactory, and leaves no doubt as to the yellow fever nature of the disorder, for we therein find that the patient experienced the peculiar pain in the head and eye so common in that fever, and the slightly inflamed condition or reddish cast of the latter organs. Besides this, he complained of considerable pain in the back and limbs; the pulse was very quick, and seldom full or tense; on the second or third day, there was a lull of all the more violent symptoms; there was considerable restlessness; the mind was generally undisturbed; the eyes and surface became jaundiced; and the scene closed with black vomit.¹

The very few writers who have spoken of this fever have, as might be expected, attributed it to a foreign source. The time had not yet come when it could be admitted to originate from the baneful action of foul local exhalations. But, as usual, opinion differed as to the mode of its introduction and the place whence it came.² One tells us that a ship from the West Indies came to the sugar-house wharf, below Cedar Street; three men were landed from her, and died of a contagious fever in the neighbourhood—and from thence the disease spread; while Dr. Redman informs us that a sick sailor, from on board a vessel from the Havana, where it then raged, was brought privately, after night, to the court near the New Market—already mentioned—before the vessel had come up to town, to the house of one Leadbetter, where he soon died, and was secretly buried. The reader will not be astonished at hearing that “Leadbetter, with most of his family and many others in that court, soon after fell a sacrifice to the distemper, and that from thence it spread rapidly, first affecting the houses nearest adjoining in Front and Pine Streets.”³

¹ An Account of the Rise, Progress, and Termination of the Malignant Fever lately prevalent in Philadelphia, briefly stated from authentic documents, 1793, pp. 32, 33, 34.

² Willing, p. 10.

³ Redman, p. 21.

EPIDEMICS OF 1793, 1794, 1797.

The yellow fever prevailed to considerable extent in New York in 1791.¹ The next year it prevailed in Charleston;² but in Philadelphia nothing seems to have been heard of the disease from the year 1762 until 1793, when, after the long interval of thirty-one years—during which the city enjoyed its usual state of healthiness, and was only visited by the ordinary diseases of the seasons, and, at times, by epidemical outbreaks of the common autumnal fever, or of eruptive complaints—the disease broke out with a severity equal to that it had manifested in 1699, and, producing a havoc proportioned to the increase of population, committed thousands to the grave.

In investigating the history of this formidable epidemic, we are not, as in some of the former instances, left to conjecture as regards the true character of the disease, and obliged to content ourselves with what may be called circumstantial evidence. We are furnished with documents of far higher value relative to that subject, as well as to the origin, extension, effects, &c., of the fearful malady, than the imperfect sketches obtained from a few letter writers, whose pursuits in life enabled them to do little more than chronicle the existence or general effects of the disease, and the popular reports of the day respecting a few of the topics in question. Philadelphia, now more than a century old, had grown to be a large city, and contained a number of professional men, natives and foreigners, distinguished for brilliant talents, and extensive and solid acquirements, well prepared to investigate every circumstance connected with the history, diagnostic signs, and pathological character of the disease they were called upon to observe and combat, and adequate to the task of committing to the press the result of their observation; while there were not wanting near them persons of literary accomplishments, who, though unconnected with the profession, were well qualified to portray the course and effects of the epidemic, as well as the circumstances under which it had appeared. The result of this improved state of things is the possession, on our part, of a number of valuable medical and other documents relative to this epidemic, from which the investigator of the present day may gather information sufficiently ample and accurate to enable him to form a just view of the whole subject, and to compare the disease with similar affections as they have appeared at various times and in other situations in this or foreign countries.³ Not a few of these publications have assumed the character of standard works, and will continue to be so regarded so long as interest is taken in the subject. From their intrinsic value, and the magnitude of the events they record, the epidemic of 1793 well forms an era in the history of yellow fever; for, on examination, it will be found that, on the observations made on that occasion, is in no small measure based the earliest knowledge obtained re-

¹ See Addoms, *Inaugural Dissertation on the Malignant Fever which prevailed in New York during the months of August, September, and October in 1791*, p. 7, N. Y. 1792.

² Ramsay, ii. p. 85; Simons, p. 6.

³ Rush, Deveze, Currie, Cathrall, Nassy, Barnwell, Helmuth, Carey.

specting the phenomena, character, origin, and mode of propagation of that disease.

The interval that had elapsed since the last outbreak of the fever was so considerable, that few individuals remained who were conversant with its phenomena, and thereby able to discriminate between it and the aggravated forms of the ordinary fever of the season. From this cause, as well, probably, as from a forgetfulness of the events of former days—which, as we have seen, had been left unrecorded—neither physicians nor, generally, the public at large, could at first discover, or be made to believe, they had anything unusual to contend against. It was some time, therefore, before the fever attracted public notice. But the death of some persons of note, the appearance of symptoms of unusual character, the rapid succession and multiplicity of cases, and the mortality which ensued, opened the eyes of all, and spread universal terror.

Nothing, so far as the weather and temperature are concerned, seemed, in the minds of the physicians, or the public, to portend the occurrence of so direful a calamity. That such a sense of security should have been felt, may appear to us strange and unaccountable; for the experience of that eventful season, combined with the result of observations collected here in subsequent years, and in other places, has taught us to connect the idea of the development of the yellow fever with the existence of atmospheric peculiarities and conditions of localities, such as were noted at the time. But, at the period in question, experience had led to no knowledge of the kind; and, to the few who bore in mind that epidemics of yellow fever had existed in former days, it never occurred that the disease might be due to such causes. Hence, what would now be considered of ominous import, was overlooked by the physicians and public of the day.

Dr. Rush, who has furnished us with one of the best accounts of this epidemic, states, that the weather was moderate during December, 1792, and the January following, but became cold in February. Blossoms, he says, were universal on the fruit-trees on the first day of April. Several warm days were experienced in the month of May, and many in June; while, during the succeeding month, July, the heat was uniformly and, towards the middle, extremely great. The same author adds, that the weather, for the first two or three weeks in August, was temperate and pleasant, but became very warm and dry in the latter part of the month.¹

During the whole month of September, and the first two weeks in October, the weather was warm and unusually calm, no breeze coming to moderate the heat. No rain fell from the 25th of August to the 15th of October, except a few drops, hardly sufficient to allay the dust of the streets, on the 9th of September and 12th of October. In consequence of the drought, the crops of grain and grass were impaired, the springs and wells failed in many parts of the country, and the dust in some places was excessive.

The temperature of May, observed in the hottest part of the day, varied

¹ Rush, Med. Inq., iii. p. 41.

from 56° to 87° ; that of June, from 60° to 92° ; that of July, from 75° to 91° ; that of August, from 66° to 90° ; that of September, from 67° to 89° ; and that of October, from 37° to 80° —the mean temperature of those months being :—

	Morning.	Mid-day.
May	59.4 . . .	72.94
June	64.87 . . .	77.10
July	72.24 . . .	84.45
August	69.03 . . .	82.55
September	60.93 . . .	76.48—only 27 days.
October	44.06 . . .	62.57—30 days.

This gives an average for the months of June, July, and August, of $68^{\circ} 70'$ in the morning, and $81^{\circ} 37'$ at mid-day. Another record gives an average, at 3 P. M., of $79^{\circ} 7'$ for June; $84^{\circ} 3'$ for July; and $82^{\circ} 7'$ for August; the average of the three months being $82^{\circ} 2'$.¹ A different set of observations, again, made at sunrise, 2 and 10 P. M., gave for May 62° , June 76 , July 81 , August 74 , September 70 , and October 64 ; the average of the whole period being $71^{\circ} 2'$, and that of June, July, and August, 77° .²

But whichever of these computations we may adopt, the result exhibits a degree of heat exceeding that ordinarily observed at the same period, and which, when viewed in connection with other concomitant circumstances, is well calculated to account, in the minds of etiologists of the present day, for the appearance of the disease, or at least to render its occurrence less extraordinary.

The register of the weather shows how little the air was agitated by winds during a considerable portion of the season. “In vain,” says Dr. Rush, “were changes in the moon expected to alter the state of the air. The light of the morning mocked the hopes that were raised by a cloudy sky in the evening. The sun ceased to be viewed with pleasure.”³ In another place (p. 7), the same writer says: “There was something in the heat and drought of the summer months which was uncommon in their influence upon the human body. Labourers everywhere gave out (to use the country phrase) in harvest, and frequently, too, when the mercury in Fahrenheit’s thermometer was under 84° . It was ascribed by the country people to the calmness of the weather, which left the sweat produced by heat and labour to dry slowly upon the body.”

The condition of the localities where the disease broke out and prevailed, was, at the time, far from being of the kind desirable to secure public health. Many portions of them were in the most impure state. The wharves and dock were more or less filthy. Dr. Rush and others laid great stress on a quantity of damaged coffee which was exposed, during the latter part of July, in a place (on a wharf, and in the adjoining dock) and under circumstances which favoured its decomposition. “Its smell was highly putrid and offen-

¹ Cadwallader Evans, *Eclectic Repertory*, vii. p. 428.

² Peirce, *Meteorological Account of the Weather in Philadelphia from 1790 to 1847*, pp. 87–192.

³ *Account of the Fever of 1793*, p. 108.

sive, insomuch that the inhabitants of the houses in Water and Front Streets, who were near to it, were obliged, in the hottest weather, to exclude it by shutting the doors and windows. Even persons, who only walked along those streets complained of intolerable fetor, which, upon inquiry, was constantly traced to the putrid coffee."¹

Besides this coffee, there was a large amount of putrid herbs and decomposed animal and vegetable substances. The narrow streets and alleys near the wharves, as, indeed, in many other parts, were in a foul state, and the gutters almost everywhere were sadly neglected.

In the February of this year, the mumps had prevailed somewhat extensively, and there appeared also many cases of scarlatina anginosa. The same diseases continued to show themselves during March, when there occurred a few pleurisies. In April, the scarlatina continued to prevail; it assumed a character of great violence in the commencement of July. Cholera morbus and remitting fever now made their appearance, and were very common in the first weeks of August. Dysentery, at the same time, was not unfrequent. In the latter end of the same month, the influenza spread extensively.² But neither this disease nor the scarlatina, which continued to prevail among children in different parts of the city, caused a great mortality. All these diseases gave way, however, soon after the appearance of yellow fever, so that few other complaints were encountered during the progress of the epidemic; when they did appear, they were generally more or less modified by it.

The disease made its appearance in its usual garb, about the middle of August. Several cases of fever of an aggravated character had manifested themselves from the commencement of the month, without, however, exciting, as already mentioned, the least apprehension of the existence of a malignant or yellow fever in the city. Sporadic cases of common bilious fever were not unfrequently seen, in which the disease put on symptoms of great malignity and terminated fatally in a few days, and now and then with yellowness of skin before or immediately after death. Dr. Rush says: "The report of a malignant and mortal fever being in town spread in every direction, but it did not gain universal credit. Some of those physicians who had not seen patients in it, denied that any such fever existed, and asserted (though its mortality was not denied) that it was nothing but the common annual remittent of the city. Many of the citizens joined the physicians in endeavouring to discredit the account I had given of this fever, and, for awhile, it was treated with ridicule or contempt. Indignation, in some instances, was excited against me."³ But, from about the 18th of the month, the rapid succession of fatal cases, attended with phenomena unusual in the common remittent of the season, left no doubt as to the true nature of the prevailing disease. The first official notice of the latter appeared on the 22d of August, when the mayor addressed a letter to the city commissioners,⁴ and, after ac-

¹ Account, &c., pp. 153-4; Med. Inq., iii. p. 109.

² Rush, pp. 4, 6, &c.; Barnwell, p. 367.

³ Account, &c., pp. 14, 15; Works, iii. p. 44.

⁴ Carey, p. 21.

quainting them with the state of the city, issued most peremptory orders to have the streets properly cleaned and purified, and all the filth immediately removed. These orders were repeated on the 27th, and similar ones given to the clerks of the market.

The disease, thus established, spread more or less extensively during the remainder of August and the whole of September. It attained its culminating point in the early part of October; decreased sensibly from the 15th of that month; was checked through the effect of cold and frost, which succeeded to refreshing showers of rain; but did not cease entirely until the close of the first week in November.

During these few months, the disease gradually invaded a considerable extent of the city. Making its first appearance in Water Street, between Mulberry and Sassafras, it for some time confined its ravages to that street—almost every case being traced to its original source. From thence it extended northwardly up Water Street to Vine. Front Street, within the above limits, was next attacked, whence it spread in the parallel streets and up those running east and west. In the progress of a short time (after the 15th of September), it prevailed in most quarters of the city—every street appearing to be charged with miasmata; attacked with considerable severity the suburb of Southwark, and broke out almost simultaneously in the district of Kensington, at that time a separate village. It did not escape observation that, during the whole course of the epidemic, the greater number of cases occurred in the vicinity of the Delaware River; that there, also, as in close alleys and small streets, the disease assumed its most aggravated form, and proved most usually fatal; and that its severity lessened in its progress westward and towards the districts.¹

“We remarked,” says Dr. Barnwell (p. 374), “all through this disease, that it was much milder in the suburbs than in the middle of the city—especially to the southward and westward it was less fatal; and in those alleys and ill-aired streets, it was much more dangerous, and also more general, than where there was a more open exposure and free air.”

This is not the place to enter into any details respecting the phenomena which the disease presented during this fearful epidemic. At a subsequent period, the subject of the symptomatology of the fever, as it has appeared in the city at various periods, will engage our attention fully, and enough will then be said to render any separate description useless here. If a different course has been pursued in reference to the former visitations, it was simply because, the accounts handed down to us being imperfect and unsatisfactory in a professional point of view, it became important to lay before the reader all the facts and circumstances we could gather, with a view to arrive at a correct knowledge of the true nature of the disease. As regards the epidemic of 1793, however, and those which followed, the same difficulty does not exist. Full and accurate descriptions of the symptoms have been laid before the

¹ Minutes of Proceedings of the Committee of Citizens, &c., on Fever of 1793, pp. 127, 128; Carey, p. 3.

public, and from them it will be easy to exhibit a portrait of the disease; not only to exhibit the latter in the various modifications it has presented in the same and at different seasons, but to establish a comparison between it and the corresponding disease, as it has been noticed in other cities of the Union and in foreign countries. Let it suffice to state in this place that the fever of 1793 presented that appearance of the skin from which it derives its usual appellation; the sudden intermission or cessation of the febrile symptoms on the second or third day; the ejection of black coffee-grounds from the stomach; in a word, the greater if not all the phenomena which characterized the fever of Charleston, as described by Lining and Moultrie; and of the West Indies, as portrayed in writings familiar to the physicians of the time—of Towne, Warren, Williams, Desportes, Desperriere, Maekittrick, Wright, Hillary, Moseley, Hume, Dazille, &c.

During the few months of its prevalence, the disease occasioned a mortality well calculated to excite alarm and dismay among the inhabitants of the ill-fated city, as well as to arouse the sympathies of the whole country. From the 1st of August to the 10th of November, the number of deaths in the city amounted to more than four thousand. According to Dr. Currie (p. 3), it did not fall short of 4,040. Dr. Rush and others stated it at 4,044 (pp. 129, 130; iii. p. 78), distributed as follows: August 325, September 1,442, October 1,976, and November 118.¹ In the course of the 101 days the epidemic lasted, the daily mortality, in 24 hours, reached between 40 and 50 twice; between 50 and 60 nine times; between 60 and 70 eight times; between 70 and 80 six times; between 80 and 90 seven times; between 90 and 100 three times; while it exceeded 100 on four occasions, all in October—102 on the 9th, 119 on the 11th, 111 on the 12th, and 104 on the 13th.

Many of the deaths which occurred in August arose from ordinary diseases; but, after the epidemic had completely established its empire, the very large proportion of deaths—all, it is supposed, but a few hundred—were occasioned by the fever.

A committee was appointed to aid the guardians of the poor in performing their duties towards the sick. This committee ordered an enumeration to be made of the inhabitants and of the houses within the city and suburbs.² The result of this enumeration was, that the city, and districts of Southwark and the Northern Liberties, contained, at the time, 6,327 houses, with a population of 36,811. The mortality in these houses amounted to 3,293. This, when added to the former sum, gives a total of 40,144 as the population at the commencement of the epidemic. Should this be correct, the loss was in the proportion of 1 in 10 of the whole number of inhabitants. It is to be remarked, however, that the census of 1790 gives a total of over 40,000, which, at the usual rate of increase, would raise the population of 1793 to more than 50,000. Admitting this to be a true estimate, the mortality would be reduced to 1 in 12.13 of the whole number.

¹ Minutes of the Proceedings, Philad. 1794.

² Seybert, Statistics of Philadelphia, p. 16; Minutes of Proceedings, &c.

The proportion of houses, population, and deaths, in the city and suburbs, was, according to the above-mentioned enumeration, as follows:—

	Houses.	Population.	Deaths.	Proportion.
City	4,222	26,146	2,223	1 in 11.31
Southwark	981	6,545	527	1 in 12.4
Northern Liberties	1,114	7,450	543	1 in 13.72

This mortality, great as it appears by the above calculation, is rendered still greater when we bear in mind that, at the time of the enumeration, 2,728 houses were closed, and not less than 12,196 of the inhabitants (Carey says 17,000) had fled the city, and sought refuge in the purer air of the country, or been removed there by the public authorities.

As to the number of individuals who suffered from the disease, and the proportion of deaths to the number attacked, nothing satisfactory has reached us. Dr. Currie (p. 3) mentions that, “at one period, there appears to have been more than 8,000 persons confined by it at the same time;” and we find that, at the Bushhill Hospital, 807 sick were admitted, of which number 448, or more than one-half, died. At a future time this subject will be resumed, when reasons will be given for believing that the proportion was about 1 in 3, and that the number attacked amounted to near 11,000.

On the 25th of August, the College of Physicians was convened to take into consideration the nature of the disorder, and the means of checking its progress. The result was an address, signed by the presiding officer and the secretary, recommending the public to avoid all unnecessary intercourse with the infected, to place marks on the doors and windows of the houses containing persons affected with the disease, and to pay the strictest regard to cleanliness and the airing of their rooms. The College further recommended the public to establish a large and airy hospital in the neighbourhood of the city, for the reception of such poor persons as could not be accommodated with the above advantage in private houses; also, to stop the tolling of the bells; to bury such persons as died of the fever in carriages, and in as private a manner as possible; and to keep the streets and wharves clean. They further remarked that, “as the contagion of the disease may be taken into the body, and pass out of it without producing the fever, unless it be rendered active by some active cause,” the following means would be necessary for the purpose of guarding against this effect: to avoid all fatigue of body and mind, and standing or sitting in the sun in a current of air, or in the evening; to accommodate the dress to the weather, and to exceed rather in warm than in cool clothing; and, while avoiding intemperance, to make moderate use of fermented liquors, such as wine, beer, and cider. They likewise declared their opinion, that fires in the streets were dangerous, if not an ineffectual means of checking the fever, and that greater advantage was to be expected from the burning of gunpowder. The benefits of vinegar and camphor, they added, were confined chiefly to infected rooms, and they could not be too often used on handkerchiefs, or in smelling-bottles, by persons attending the sick. (*Carey*, p. 22.)

The official announcement of the existence of a fatal and spreading epi-

demic, by the city authorities, created no small alarm among the inhabitants. Incredulity in the minds of physicians and the public ceased, and gave place to a sense of danger; and means were devised by the latter to shield themselves from the impending evil. But great as this alarm may have been, it sinks into almost insignificance when compared to what it became on the publication of the address of the College of Physicians. For no sooner was the pestilential character of the disease positively affirmed by a body of men who, from the nature of their pursuits, were naturally supposed capable of forming a correct opinion on the subject—no sooner were all the recommendations conveyed in that document promulgated, than alarm changed into terror; consternation was carried beyond all bounds; dismay and fear were visible on almost every countenance. The greater number of those who could by any means escape from the city, lost no time in effecting that object. “So great was the general terror, that, for some weeks, carts, wagons, coaches, gigs, and chairs, were almost constantly transporting families and furniture to the country in every direction. Many people shut up their houses wholly; others left servants to take care of them. Business became extremely dull. Mechanics and artists were unemployed; and the streets wore the appearance of gloom and melancholy.” (*Carey*, p. 21.)

The chroniclers of the events of the times, Rush, Carey, Deveze, Nassy, Volney, and Helmuth, furnish us with a dismal picture of the moral condition of the afflicted city, from which I cannot glean more than a few passages.

Many of those who remained being afraid to walk the streets, lest they might meet with individuals impregnated with the seeds of the pestilential infection, shut themselves up in their houses. Thousands, of all sexes and ages, resorted to the smoke of tobacco as a preventive against the contagion. Some chewed garlic, and wore it about their persons. Others, carrying their caution to a still greater length, provided themselves with new lancets, to be used on themselves or their families, in order to avoid thereby contamination from the instruments of physicians or bleeders. Many houses, as we are informed, were scarcely a moment in the day free from the smell of gunpowder, burned tobacco, nitre sprinkled with vinegar, &c. Churches were deserted, or wholly closed—as also the Exchange, City Library, and public offices—while the greater number of public papers were discontinued. Never were the rooms of Philadelphia oftener and more thoroughly purified, scoured, and whitewashed, than on this frightful occasion. Few went abroad without protecting themselves by the constant application to their olfactory nerves of handkerchiefs, or sponges, impregnated with vinegar or camphor. Some were found carrying pieces of tarred rope in their hands or pockets, or camphor bags tied around their necks. Many never walked on the footpath, but took to the middle of the street to avoid being infected in passing houses where death from the disease had occurred. Acquaintances and friends avoided each other in the streets, and contented themselves with a cold nod. Shaking of hands fell into disuse, and cases of persons shrinking back with affright at even the offer of the hand are recorded. A craped hat, or any other token of mourning, was carefully shunned; and many persons valued

themselves on the skill and address with which they gained the windward of those they met, and all uniformly and hastily shifted their course at the sight of a hearse. (*Carey*.) "The streets," says Dr. Rush, "everywhere discovered marks of the distress of the city. In walking for many hundred yards, few persons were met, except such as were in quest of a physician or bleeder, or the men who buried the dead. The hearse alone kept up the remembrance of the noise of carriages or carts in the streets. Funeral processions were laid aside. A black man, leading or driving a hearse, with a corpse on a pair of chain-wheels, with now and then half a dozen relations or friends following at a distance from it, met the eye in most of the streets of the city, at every hour of the day; while the noise of the same wheels, passing slowly over the pavements, kept alive anguish and fear in the sick and well every hour of the night."

It was impossible to find carriers of the dead, as a number of those who had at first performed that sad office were soon infected by the disease. It became necessary, in consequence, to resort to carriages for that purpose. The number of attendants at funerals was greatly diminished, and everybody retired at the approach of a hearse, and windows and doors were shut as they passed. The day, frequently, would not suffice to bury all the dead, because no assistance was to be had in digging the graves. Much of this was, therefore, done by night.

In a word, scenes similar to those we read of in the history of the disastrous plagues of Marseilles, of London, and other European cities, and of the epidemics of the middle ages, were enacted in Philadelphia during the eventful autumn of 1793. It is painful to reflect, that such were not the only points of resemblance between those pestilences and that which visited our city. Here, as elsewhere, the distresses of the times called forth, in the strongest relief, a feeling of selfishness, and not a few instances occurred in which the endearing ties of humanity were dissolved. Husbands were found who did not scruple to desert their wives at the critical moment, while the death-beds of many were unsoothed by the presence of their wives. Parents forsook their children; children forsook their parents; masters turned their servants adrift, or consigned them to the tender mercies of the hospital; and, on the other hand, some domestics abandoned humane masters at the moment of the greatest need, and, in some instances, those of affluent fortune, who had given daily employment and sustenance to hundreds, were turned over to the care of a mercenary negro, after their wives, children, friends, clerks, and servants had fled away and left them to their fate.¹

But, while such was but too frequently the case—while the feelings of humanity were loosened to a deplorable extent—it is a source of consolation that there were not wanting instances in which these same feelings manifested themselves with increased splendor, and that the calamities of the times brought forth examples of the most exalted benevolence and philanthropy. Without stopping to specify these, it may not be improper—if, indeed, it is not a duty

¹ *Carey*, p. 31; *Deveze*, preface, xii.

—to record in this place that, among the members of the medical profession, the most entire devotedness to their fellow-creatures was early and continuously displayed.¹ With a few exceptions, they remained at their post, ministering to the sick. Amid the appalling scenes of distress and destruction by which they were surrounded, the sense of self-preservation was no longer operative. Unheeding the cause of infection, to which, more than any other classes, they were exposed, they applied themselves to the performance of their laborious professional duties with a calmness and zeal worthy of all praise. In the short space of five or six weeks, exclusive of medical students, not less than ten physicians were swept away by the epidemic. Scarcely one of those that survived, or remained in the city, escaped sickness. Some were three, four, or five times confined;² and, such was the condition to which they were reduced, that Dr. Rush states that, at one time, there were but three physicians able to do duty out of their houses. Our celebrated countryman must have forgotten the French physicians, who never ceased to attend to their professional duties.

As might be readily expected, the causes, as also the mode of propagation, of the fearful fever of 1793 gave rise to much discussion. I need scarcely remark that this, like many other visitations of the kind, was attributed to the wrath of the Deity, as a punishment for the sins of the inhabitants. Ministers, mounting the pulpit, were eloquent, and waxed warm on the subject. Texts were liberally furnished to show that such punishments were not of unusual occurrence. Some of these sermons were published, for the benefit and conversion of such of the sinners as had not enjoyed the advantage of hearing them; and, therein, sins were pointed out and dwelled upon as the principal cause of the calamity, which the people have not, from that day to this, shown any disposition to amend.³

But it forms no part of my plan to enlarge on the etiological theories of such inquirers, and I must revert to matters of greater professional importance. By the majority of physicians, and the public at large, the disease

¹ See Deveze, preface, xii.

² Carey, p. 65.

³ "Philadelphia," says one of these preachers, "far exceeded most of the cities of North America in luxury and dissipation among all classes of people.

"It was Philadelphia that did not rest until the performing of theatrical exhibitions was authorized by law. It was Philadelphia that refined so much on this species of vanity, as to erect one of the largest houses on the continent for theatrical exhibitions, and engaged actors at a prodigious expense; as if one house, that existed before, were not sufficient to ruin our young people, too much neglected already. It was Philadelphia that imported from luxurious Europe the number of seventy or eighty actors and retainers of the stage, who actually arrived here exactly at the time when the fever raged with the utmost violence. It was Philadelphia that contained those parents who had given willingly three hundred dollars to obtain a perpetual right of free access, with wife and children, to the plays, in order to plunge themselves and their relatives the quicker into all kinds of dissipation, and obliterate in their hearts all taste for what is serious and useful, I will not say godly and heavenly" (p. 12).

J. Henry Helmuth (minister of the Lutheran congregation), *A Short Account of the Yellow Fever in Philadelphia. For the reflecting Christian.* 1794.

was ascribed to importation from the sickly ports of the West Indies. The College of Physicians, to which the Governor of the State applied for information respecting the origin of the disease—whether or not it was imported, and, if so, “from *what place*, at *what time*, and in *what manner* it was introduced”—gave a decision in approval of that doctrine. After affirming that no instance had ever occurred of the disease called *yellow fever* having been generated in this city, or in any other parts of the United States, but that frequent instances had occurred of its having been imported, not only into this, but into other parts of North America, and prevailing there for a certain period of time, they continued: “From the rise, progress, and nature of the malignant fever which began to prevail here about the beginning of last August, and extended itself gradually over a great part of the city, we are of opinion that this disease was imported into Philadelphia by some of the vessels which arrived in the port after the middle of July. This opinion we are further confirmed in by various accounts we have received from unquestionable authorities.” This opinion of the highest medical tribunal of the city and country—which was a mere echo of that entertained in former days relative to the epidemics already described—found numerous supporters among the members of the profession, and was almost universally adopted by the public at large. But, though the Governor requested to know from what place, at what time, and in what manner the disease had been imported, the College took no notice of any of these questions. “Public report had derived it from several islands; had chased it from ship to ship, and from shore to shore; and, finally, conveyed it at different times into the city, alternately by dead and living bodies,” and it was on these popular and fluctuating reports that the opinion of the College was based. It was reserved for after times to see the establishment and adoption, by the advocates of importation, of a fabric at once more plausible and enduring.

But, while the doctrine advocated by the College was thus supported by the majority of the physicians and the public at large, it met with opposition from several influential members of that body, and some physicians of eminence. Dr. Redman, the president of the College, Dr. Foulke, and Dr. Leib, dissented from the report contained in the letter in question; Dr. Hutchinson, the inspector of sickly vessels, in an official communication, remarked that it did not appear to be an imported disease; and by these, and Dr. Rush, the doctrine of importation was rejected, and the opinion of the local origin of the fever openly defended, on grounds which will form the subject of investigation in a subsequent part of this volume.

As regards the mode of propagation of the disease, a greater unanimity of sentiment existed at the outset of the epidemic, and for some time after. By the very large majority of the physicians and the public generally—even by those who advocated the opinion of local origin—it was thought to be transmitted by contact or intercourse with the infected; in other words, through means of contagion. But, even at that early period, physicians were found who rejected such a belief, and taught that the cause of the extension of the fever was to be sought in the diffusion, through the agency of the air,

of morbid exhalations, themselves the offspring of organic decomposition. Gradually, the doctrine of non-contagion gained converts, until, finally, the physicians of Philadelphia became divided into two almost hostile parties. From this moment may be dated the origin of the interminable dispute about contagion and non-contagion, which has continued ever since to occupy the attention of the medical profession in this and other countries.

This fatal epidemic was commemorated in verse, while some of the melancholy scenes to which it gave rise furnished materials for a striking chapter in one of the best works of a distinguished American novelist.¹

1794.—The inhabitants of Philadelphia had scarcely had time to recover from the effects of the severe calamity by which their city had been visited in 1793—sweeping off a large portion of the population, and crippling its prosperity in many ways—before they were threatened with a renewal of the same scenes. The yellow fever made its appearance the next year; but happily in a less extended and devastating form. By many, indeed, its existence at the time was doubted, and even denied. The public would not, or could not be made to believe that their city could once more be the theatre of a fatal epidemic, or troubled with numerous sporadic cases of the fever. So decided was the belief on the subject, or the determination to conceal the fact, that no record of the disease is to be met with in the newspapers, or other public documents of the time; and with the exception of a separate short essay on the occurrences of that year, for which we are indebted to Dr. Rush,² they are only incidentally and briefly noticed in a few publications, specially devoted to other and more important epidemics.³ Dr. Rush, indeed, became the object of much obloquy for having openly and early proclaimed to the city authorities, and the public at large, that a fever, similar to that from which they had so much suffered, was among them. There were not wanting physicians who joined in the clamor against that eminent man and those entertaining like views; and who, “while they daily saw and attended, or lost

¹ As Mervyn approached the city, “the tokens of its calamitous condition became more apparent. Every farm-house was filled with supplementary tenants—fugitives from home, and haunting the skirts of the road, eager to detain every passenger with inquiries after news. The passengers were numerous, for the tide of emigration was by no means exhausted. Some were on foot, bearing in their countenances the tokens of their recent terror, and filled with mournful reflections on the forlornness of their state. Few had secured to themselves an asylum; some were without the means for paying for victuals, or lodging for the coming night; others, who were not thus destitute, yet knew not whither to apply for entertainment, every house being already overstocked with inhabitants, or barring its inhospitable doors at their approach.

“Families of weeping mothers and dismayed children, attended with a few pieces of indispensable furniture, were carried in vehicles of every form. The parent or husband had perished, and the price of some movable, or the pittance handed forth by public charity, had been expended to purchase the means of retiring from this theatre of disasters, though uncertain and hopeless of accommodation in their neighbouring districts.”—*Arthur Mervyn*, by Brown, i. pp. 125, 126.

² Rush, *Fever of 1794*, Med. Inq., iii. p. 197.

³ Folwell, *Fever of 1797*, p. 3.

patients in the yellow fever, called it by less unpopular names"—alleging, that the prevailing disease was not of the nature contended for, because some who had died of it had not a sighing in the beginning, and a black vomiting in the close of the attack; and affirming, that cases in which the black vomiting and yellowness of the skin occurred, were not necessarily cases of yellow fever, inasmuch as such symptoms were not unfrequently observed in other diseases. "Notwithstanding the pains which were taken to discredit the report of the existence of the yellow fever in the city," Dr. Rush continues, "it was finally believed by many citizens, and a number of families, in consequence of it, left the city. And in spite of the harmless names of intermitting and remitting fever which were given to the disease, the bodies of persons who had died with it were conveyed to the grave, in several instances, upon a hearse, the way in which those who died of the yellow fever were buried the year before."¹

That all the cases of severe and even fatal fever which occurred in the early part of the season were of the nature contended for by Dr. Rush, is certainly more than we would now be disposed to acknowledge. Experience has shown not only in this city, but in every other part of our country subject to the disease, that the yellow fever requires for its development a concurrence of circumstances seldom, if ever, to be found with us until some time has elapsed after the period at which the first cases noted had appeared. We all know that Dr. Rush entertained peculiar views respecting the unity of febrile diseases, which must have led him—occasionally, at least—to regard as similar, fevers which others view in a different light. Indeed, it is impossible to read his account of the yellow fever of 1794, without perceiving the slight grounds upon which the early cases were pronounced to be of the nature in question. But, disposed as we may be to adopt this opinion, the proofs we possess in relation to the character of the cases which appeared later in the season are such as to remove all doubts on the subject. Besides this, we have the testimony of other physicians perfectly conversant with the fever. Dr. Deveze, than whose authority none could be higher, informs us, that the yellow fever occasionally appeared in the year in question (xix.). Dr. Monges, and other French physicians, whose knowledge of the disease was equally extensive and accurate, and whose views I had ample opportunities of ascertaining, coincided with Dr. Rush and Dr. Deveze on the subject; and we further learn that there was scarcely a citizen or physician who, three years after, did not admit of its having prevailed that year. In addition, it may be remarked that the yellow fever appeared that season in Baltimore,² New York,³ New Haven,⁴ and Charleston;⁵ from which we may infer that there existed that year an epidemic constitution of atmosphere, which was extended to this city, though in a feebler degree, and which, had it been aided

¹ Rush, *op. cit.*, p. 201.

² Drysdale, *Med. Mus.*, i. p. 2.

³ Rush, *Ibid.*, iii. 218.

⁴ Two letters relative to the yellow fever as it appeared in New Haven in the year 1794, by Dr. Monson, Webster collection, 171, &c.

⁵ Ramsay, *Hist. of South Carolina*, ii. p. 85; Simons, p. 6.

by strong, remote and exciting causes, would doubtless have elicited the development of the fever on a larger scale.

The precise period at which the true yellow fever made its appearance this year cannot now be ascertained. There is reason to believe, however, that cases of the disease began to show themselves in the month of August—about the time it broke out in Baltimore and elsewhere. From this period it continued to manifest itself until checked by the cold weather of the latter part of autumn.

It made its appearance in the usual localities—prevailing in Water Street, between Market and Walnut, and extending westwardly in various directions. Nor is it possible, at the present time, to ascertain the number of cases of the fever which occurred during the course of its prevalence, or of the mortality it occasioned. No record was kept on either topic, and I can find nothing in Dr. Rush's essay, or other publications, from which a satisfactory estimate can be made. Like the preceding epidemic, that of 1794 appeared under the influence of a high temperature. According to the observations of Mr. Cadwallader Evans, June presented an average of 75.6; July, 80.4; and August, 81.7; with a mean for the three months of 79.2.¹

Like the fever of the preceding year, that of 1794 was ascribed by many to importation from abroad. A vessel which arrived from St. Mark on the 25th of August was fixed upon as the instrument of introduction, and on the strength of this supposition, physicians who otherwise would have been in doubt as to the existence of the fever, now admitted it to be in town. But by some a different sentiment was entertained relative to the origin of the disease; for Dr. Rush, and many others, ascribed it, as they had done before, to the exhalations from the wharves, gutters, and stagnant ponds. That the former were in a fit state to generate morbid exhalations, will easily be inferred from what has been already stated; and in reference to the neglected state of the gutters, and other sources of stagnating filth, we have the fullest evidence. It was not to be expected that the citizens of Philadelphia, who had an interest in rejecting the proofs of the generation of the disease in their city, or who could not be made to comprehend how a malignant fever might arise from exhalations which ordinarily remained harmless, or who were captivated by the more striking idea of foreign importation, should devote much attention, time, and expense to the introduction and enforcement of regulations necessary to prevent an effect, which, to them, was more than problematical. Hence, the summer and autumn of 1794 saw the gutters, and other sources of filth, much in the same state they were in the year preceding.

During the course of the summer and autumn of the two succeeding years, the yellow fever appeared sporadically in Philadelphia, but not to a sufficient extent to create much alarm, or deserve special notice in this place.² It may be remarked that, in the first of these years, the disease prevailed extensively in the city of New York, where it carried off, in a few weeks, upward of

¹ Eclectic Reprint, vii. p. 428.

² An Account of Sporadic Cases of Bilious Yellow Fever in Philadelphia in the years 1795 and 1796. By Dr. Rush. Med. Inq., iii. p. 241.

seven hundred persons; and also, with great mortality, in the cities of Norfolk (Virginia), Charleston, &c. In the next year, it broke out again in Charleston, also at Wilmington, N. C., and at Newburyport, Mass. The origin of the fever in most of these places became a subject of more or less contention; but the opinion of its being due to local sources of infection was warmly defended in New York and Norfolk, was scarcely, if at all, opposed in Charleston, where it finally prevailed almost universally. The fact of the prevalence of the fever in the epidemic form in other cities of the Union, and its appearance, though only in isolated cases, within the limits of Philadelphia, seemed, according to some, to indicate the existence over the whole, or at least an extensive portion of the country, of an epidemic constitution of atmosphere, which, not meeting here with the same amount of local impurities, and consequent febrific exhalations, and unaided by the ordinary exciting causes of the disease, manifested its presence by the production of only a limited number of attacks; while others, denying the yellow fever nature of these sporadic cases, ascribed the exemption to the non-arrival of infected vessels from the West Indies. Whatever truth there may be in either of these explanations, it was matter of observation that, while the local sources of infection in all probability existed in 1795-6 much as they had done in 1793, the meteorological conditions were not such as usually prevail in yellow fever seasons. In 1795, April was wet and cold; May was alternately wet, cool, and warm; while in July, though some days were wet (17, 20, 21, 22), the rest of the month was otherwise. At the close of the month, and beginning of August, rain fell alternately. In 1796, May and June were uncommonly hot; but July, instead of being hot and dry, was very wet, from a repetition of heavy showers. Peirce,¹ from whose meteorological records the above is taken, gives as the medium temperature of the three months, June, July, August, 1794, $71^{\circ} 3'$. In 1796, it was $71^{\circ} 7'$. In 1793, it was, agreeable to the same observer, 77° ; and we shall see that, in other epidemic periods, it was equally high.

1797.—Very different was the extent of the prevalence of yellow fever in the year succeeding to that last mentioned. On this occasion, it once more assumed the character of an epidemic, less extensive and fatal, doubtless, than that of 1793 and some that followed, but of sufficient severity to merit a somewhat detailed notice. It was described by Dr. Rush,² Dr. Pascalis,³ and others,⁴ whose publications on the subject will amply repay a perusal.

The spring in that year was tardy, and the weather in June and July was dry—little rain falling during those months. In the early part of August, from the 2d to the 9th, the weather was wet, but it afterwards once more became dry. The thermometer, during the course of the summer, did not range as high as it had done in 1793, but high enough to produce a mean

¹ A Meteorological Account of the Weather in Philadelphia, &c. Phil. 1847.

² An Account of the Yellow Fever as it appeared in Philadelphia in 1797. (Ed. of 1798.) Also, Med. Inq., iv. p. 3.

³ An Account of the Contagious Epidemic Yellow Fever, &c., of 1797.

⁴ Short History of the Yellow Fever that broke out in Philadelphia in 1797. By R. Folwell.

temperature, at 3 o'clock, in June, July, and August, of 80.7° .¹ The average heat in August, September, and October, was—

	Morning.	Mid-day.
August	69.10°	74.25°
September	57.80	67.67
October	44.06	57.42

The mean temperature of these months, at the former period, was 56.93.

The disease prevailed about Pine Street wharf, in Water and Penn Streets, and also, and principally, in the suburbs of Southwark and Kensington. While these spots were severely visited with the disease, the city, both along the wharves and at a distance from them, remained healthy from Walnut to Vine Streets. Some cases, it is true, occurred in the city proper in the course of September and October; but most of these were readily traced to the above sources. During its prevalence, other diseases did not disappear. Dysenteries, cholera morbus, scarlatina, and mumps were encountered in July, and bilious remittent fevers were not uncommon.

As usual, the disease did not commence before the close of July. The first unequivocal cases occurred about the first week of August. It prevailed during the remainder of that month, as, also, during September and October, and was—as in most other instances—checked by the advent of winter frost, which that year occurred earlier than it had done in 1793—*i. e.* about the middle of October.

The condition of the localities where the disease appeared and was mostly prevalent, differed in nothing from what it was in former visitations. The Academy of Medicine, in a letter to the governor, called attention to the putrid exhalations from the gutters, streets, ponds, and marshy grounds of the neighbourhood of the city, and traced to these sources most of the cases which occurred in the city and at Kensington Bridge. It also indicated exhalations issuing from vessels that had arrived from foreign ports, particularly the *Sam* from Marseilles.² Dr. Coxe depicted the offensive state of Kensington, in a document of interest, published by Dr. Rush in his account of the fever of that year (p. 82). “Sinks,” says Dr. Pascalis, “then existing to receive the water of the gutters in places where there was not a sufficient declivity to carry it off, were in a bad condition, and emitted a very disagreeable stench. The same may be said as regards the dock, sewers, &c. They all exhaled the most noxious effluvia; for dead animals, and every kind of nauseous substances, are thrown into them, and remain there until they become putrefied.”³

Though the disease was not so prevalent as it had been in 1793; though it spread over a less extensive surface, and attacked within the range of its influence a smaller proportion of a population greatly thinned by emigration, yet the number of cases must have been considerable, judging from the mortality occasioned by it. Dr. Rush (p. 18) estimates that mortality, in August, September, and October, at between one thousand and eleven hundred; while Dr.

¹ Cadwallader Evans, *Eclect. Rept.*, vii. p. 428.

² Rush, *Fever of 1797*, see p. 48.

³ Pascalis, p. 95.

Pascalis, who obtained his information from the records of the Board of Health, computes it at about 1,300 (p. viii.).¹ The epidemic appears to have reached its height, not in October—as was the case in 1793—but in September; the mortality in that month being twice as large as it was in the following, and nearly as great as during August, as may be seen by the following statement, which I derive from Ruston:²—

August	303
September	579
October	386
November	24
										<hr/> 1,292

It is to be remarked, in explanation of the comparatively greater mortality in September, and the rapid decrease which took place in that respect during the following month, that the severe frost, by which the disease is usually checked, occurred, as already stated, earlier than it had done in preceding sickly seasons, and thus, probably, prevented the epidemic from following the same course it had followed before.

As might have been readily foreseen, the intelligence of the existence of an epidemic created considerable alarm in all parts of the city. The sufferings of 1793 being fresh in the recollection of the inhabitants, and experience not having yet taught them—or their fears closing their eyes to the fact—that the disease limited its sphere of operation to a comparatively small portion of the city, and the belief in the transmission of the fever by personal contact being then almost general, the alarm spread in every direction—even to places the most remote from the infected district, and the least likely to be affected. “The terror of the citizens,” says Dr. Rush, “for awhile, was very great. Rumours of an opposite and contradictory nature, of the increase and mortality of the fever, were in constant circulation. A stoppage was put to business, and it was computed that about two-thirds of the inhabitants left the city.” Dr. Deveze, in a letter to the Governor of the State, gives an account of the state of the public mind on the subject not less gloomy than that he had noticed during the epidemic of 1793 (p. xxv.).

As on a former occasion, the disease bore heavily on the members of the medical profession. During the course of the epidemic, as we learn from Dr. Rush, not more than three or four and twenty practitioners attended patients in the disease. Of this small number, nine—several of whom were gentlemen of the most respectable character—fell victims to its ravages; while, of the survivors, eight were affected with more or less severity.

The yellow fever of 1797, like that which had prevailed before, was very generally ascribed to importation from the sickly ports of the West Indies. By the Governor of the State, a letter was addressed to the College of Physicians, requesting information as to the origin, progress, and nature of the fever and the means of preventing its return. Similar information was also

¹ Folwell at 988, p. 64.

² A Collection of Facts, &c., on the Nature, Causes, and Cure of the Yellow Fever, p. 64.

solicited from such practitioners as were not members of the College. Faithful to the views it had adopted and expounded in reference to the epidemic of 1793, the College, in an elaborate memorial to the legislature, asserted that the fever had been imported from abroad, and traced it to two vessels—the one from the Havana, the other from Port au Prince; and, refusing to recognize in local exhalations any other than influences of a secondary or accessory character, contented itself with recommending, as a most effectual means of preventing the recurrence of the disease, a more stringent system of quarantine regulations.¹

But, while the doctrine of importation was thus earnestly and strenuously advocated by the College of Physicians, views of an opposite kind, which, since '93, had gained proselytes, were as earnestly supported by a large number of physicians; thirteen of whom, in two letters to the Governor, the one in their private capacity, and the other as members of the "Academy of Medicine," discarded the idea of importation, and advocated decidedly the local origin of the disease, referring it to putrid exhalations, and to the foul air of two ships that had arrived from Marseilles and Hamburgh, and had, while discharging their cargoes, infected the vicinity of the wharves; the former at the foot of Pine Street, and the other in Kensington. They enumerated all the common sources of malignant fevers, and recommended their removal from the city, as the most effectual method of guarding against a renewal of the disease.² Dr. Deveze, too, as already stated, addressed a letter to the Governor, in which he reiterated the views he had expressed and published in 1794, respecting the local origin and non-contagious character of the fever (xxii., *note*).

As to the mode of propagation of the fever, after it had become established, a greater unanimity of opinion existed. The doctrine of contagion continued still to prevail almost universally among physicians and the public at large. As was the case in 1793, those who attributed the disease to a foreign source very naturally regarded it as spreading through the effect of direct or indirect contact; and, among such as denied importation, there was scarcely one, Dr. Caldwell—who that year changed the opinion he had formerly upheld—Dr. Deveze, and other French physicians excepted, but what adopted the same views. The time was near at hand, however, when matters were to take a different turn.

On the 11th of August, the Governor of the State, influenced by the idea that the disease was contagious and the result of importation, announced by proclamation that a pestilential fever existed in the West Indies, and enjoined that every vessel from the islands, from New Orleans, from any French, Dutch, or Spanish ports on the main, should perform quarantine for five days at the health office or State Island, or for such longer time as the resident physician and officers of that office should advise.³ In answer to a letter from the Governor, the College of Physicians, through their president, Dr. Red-

¹ See Currie on Bilious Fever, p. 225. Rush, Fever of 1797, p. 58.

² Proofs of the Origin of the Yellow Fever in Philadelphia and Kensington, in the year 1797, from Domestic Exhalations, &c., by Acad. of Med., p. 2, &c. ³ Folwell, p. 4.

man, stated that such a disease had appeared; and, in another communication to him, gave their opinion of the best mode of averting the threatening calamity, by first prescribing the methods for preventing the spreading of contagious diseases when introduced; and, secondly, for guarding against their introduction.¹ On the 21st of August, the Inspectors of the Board of Health, in a public address, stated that the infection was so limited in extent that, by proper exertions, it might, in their opinion, be entirely removed. With the approval of the Governor, they directed that every person infected, and whose case would admit of removal, should be conveyed to a proper situation distant from Philadelphia; that the adjoining inhabitants should immediately leave their premises; that a yellow flag should be placed to the houses containing, or which had recently contained, the sick; that no person, the needful attendants excepted, should enter any such house; and that physicians should give notice of such contagious cases as came under their observation. And it was further announced that a number of houses and tents had been prepared for accommodating the healthy people who might leave the city.²

It was also ordered that citizens in general, and especially those keeping lodging-houses for sailors and passengers from vessels, should give notice to the Board of Health of persons seized with indisposition having the appearance of yellow fever, to the effect of their being removed to the City Hospital. (*Ib.*, p. 6.) When the disease was ascertained to exist in several houses near each other in any part of the city, it was directed that all the neighbouring families who had escaped the infection should be removed; and that all communication between the infected families and the city be suspended, by preventing any persons, except those whose visits were essentially necessary to the sick previous to their removal, from entering into that part of the town. For this purpose, mercantile business had to be suspended there, and the vessels removed from the adjoining wharves; while all suspected wharves and houses, particularly such as had been occupied by the sick, were directed to be purified in the manner recommended by the College. (*Ib.*, p. 10.) On the 23d of August, the Governor issued a proclamation, wherein he ordered, among other things, that all communication should be cut off with the wharves, houses, and inhabitants situated between Spruce and South Streets; to extend from the river to the west side of Front Street. Necessary attendants only were to be admitted within this spot. Poles and fences were to be placed across the streets and avenues leading to it. Yellow flags were to be placed at the corner of each of the streets leading to the infected district.

EPIDEMICS OF 1798, 1799, 1802, AND 1803.

Severely as the city of Philadelphia had been visited in 1797, and especially in the memorable autumn of 1793, it pleased Providence to put its inhabitants, in 1798, to a trial of perhaps still greater magnitude. The epidemic of that year, happily the last of the kind I shall have to record, was the most extensive, in point of malignity and mortality, of any that had yet occurred in this city, and may, in these respects, be placed on a parallel with

¹ Facts and Obs. by College, p. 8.

² Folwell, p. 11.

even those by which portions of Spain, and our own city of New Orleans, have on several occasions been devastated. It afforded the last great manifestation of the epidemic constitution of the atmosphere—so far, at least, as this city is concerned—which had evidently prevailed during several years, and given rise to so much distress, and which, at the season under consideration, gave evidence of its existence and wide diffusion by the elicitation of the disease not only in Philadelphia, but also in Boston, New York, Portsmouth (N. H.), Newport (R. I.), New London, Wilmington (D.), Charleston, in most of which it assumed a severe form. True it is, as we shall see, the disease broke out the next year, and has done so at subsequent periods; but, on those occasions, it appeared in a less violent epidemic garb, or showed itself only sporadically.

The spring of 1798 assumed, at the outset, a promising aspect. There was plentiful rain, and vegetation commenced early. But a change took place about the middle of April, when a snow storm, succeeded by very cold weather, injured the sprouting leaves and blossoms. The intense heat of summer came on suddenly; the thermometer of Fahrenheit rising, on the 1st of May, as high as 84° . However, this spell was of short duration, and during that month and June, the weather, though warm, was variable, and at times attended with frost. In July, the heat became considerable, and continued so throughout, excepting towards the middle of the month, when the temperature suddenly lowered in consequence of a hail storm which occurred at some distance from the city. Towards the close of the month, the heat again became extreme, the thermometer ranging from 88° to 92° , and rising as high as 96° . Throughout August and September, the weather was marked by high temperature. During the latter part of May, and in June, part of July, and throughout August and September, the weather was very dry; and, as a consequence of the drought, whole fields were burnt up by the sun, and the crops of hay seriously injured.

The mean temperature of the season was, for

	Morning.	Mid-day.
June	66.47	76.83
July	67.61	79.77
August	73.90	83.23
September	59.50	72.43
October	48.26	60.61
November	29.53	42.06

It is well to remark, that the mean temperature of the first three months of that series—the most important in reference to the development or diffusion of the cause of the yellow fever—was not lower at mid-day than $79^{\circ} 94'$, an elevation greater by several degrees than that observed in other yellow fever seasons, but below that of 1793, when, as we have seen, it rose to $81^{\circ} 37'$. During the season, the thermometer rose forty-one times at mid-day (3 o'clock) above 80° .¹

¹ Caldwell, Fever of 1805, p. 17. Agreeable to the observations of Cadwallader Evans, the mean temperature of June was 77° , of July 82° , and August $86^{\circ} 5'$, giving an average, for the period, of $81^{\circ} 8'$. (*Eclectic. Repert.*, vii. p. 428.)

The localities where the fever made its appearance and prevailed, were in no better condition, in point of cleanliness, than those that had been the theatre of its ravages at former periods. The sinks and sewers were, as heretofore, filled with putrefying materials, and emitted the same effluvia. Condie and Folwell, though far from being friendly to the doctrine of local origin, call attention, nevertheless, to the impure condition of the city. They speak of many vacancies on the banks of the river, covered with a thick bed of miry filth; of the wharves becoming filled up with impure materials from the adjoining streets, and emitting, at low water, a very offensive stench. They specially refer to Pegg's Run, with its wide and miry bottom, and with its banks unimproved, and rendered still more injurious by being made a receptacle for the offals from slaughter-houses, tanyards, &c.; of the offensive condition of privies in thickly inhabited places; of the entire want of these in Water Street, and the offensive smell issuing from them everywhere during moist, calm, and sultry weather—particularly towards the close of summer. The Academy of Medicine, in like manner, called attention to the subject, dwelling particularly on the “putrid exhalations of alleys, and gutters, and docks, and of the stagnating waters in the neighbourhood of the city.”¹

The disease began this year early in August. In the July preceeding, and especially during the very hot weather that occurred in that month, cholera morbus prevailed to a considerable extent among children, and occasioned great mortality. Dysentery, also, was rife; and towards the middle and the close of the month, several cases of bilious fever of a highly malignant grade occurred in the western part of the city; and, in general, proved fatal. Everything, indeed—continuity of high temperature, dryness of weather, accumulation of sources of morbid exhalations, and the nature of the prevailing diseases, as well as the appearance, about the same time, of the fever in other parts of the Union, seemed to presage a sickly season; yet it was not until the 7th of August that the disease was officially announced by the Board of Health, on information communicated by the College of Physicians. Assuming rapidly a fearful character of malignity, and spreading far and wide, it prevailed from that period through the rest of the summer, and during the whole of autumn. From the 1st of October the fever gradually abated, probably from a deficiency of subjects; for many families having, in consequence of great diminution in the mortality, returned to their homes, the disease broke out with renewed violence, and continued to prevail until checked by the advent of frost. On the 1st of November its total extinction was officially announced by the Board of Health. (*Currie*, p. 136.)

As in most other epidemics, the principal seat of the disease, on this occasion, was the river side and the adjoining streets. It began about Spruce and Walnut Streets, and the wharves; but after a short time, and before the close of August, spread in nearly every part of the city. About the same time it appeared, as it had done before, in the district of Southwark, and the village of Kensington. On this occasion, and this only, the disease pene-

¹ Condie and Folwell, p. 50; Currie, p. 12; Rush, *Med. Inq.*, iv. 48.

trated into the jail, situate at the corner of Sixth and Walnut Streets,¹ and even into the Pennsylvania Hospital, in Pine, between Eighth and Ninth Streets.²

The pathognomonic symptoms of the fever were the same this season as they had been on former occasions. The disease appeared, however, to possess a greater malignancy of character, and presented some peculiarities of phenomena, a notice of which will find a more appropriate place in another part of this volume.

The mortality in this memorable epidemic was larger—not in the aggregate, but in proportion to the number of individuals attacked, or those who continued exposed to the infection—than it had been during the autumn of 1793. The whole number of deaths reported to the Board of Health from the beginning of August to the close of the epidemic, was 3,645. But it is supposed that the fatal cases that occurred in the country would, if fully ascertained, swell the number to four thousand. Of these reported deaths, 514 occurred in the City or Fever Hospital, and 3,131 in the city and liberties. From the following table it would appear that the disease attained—as was the case in 1797—its highest intensity in September:—

August (from 8th)	626
September	2,004
October	943
From 1st to 5th November	72 ³

When we take into consideration the state of the population at the time of this epidemic, this mortality must, as that which had occurred five years before, appear very large. The whole number of inhabitants in the city and liberties in 1800 amounted, as we have seen, to somewhat more than 70,000; but by the historians of the epidemic, the number, in 1798, was not estimated at more than 60,000.⁴ Assuming the latter as the probable amount, it would follow that there died, during three months, 1 in 16.48 of the entire population. This proportion, which, under all circumstances, would appear large, must cease to be so considered when we learn the state of public mind from an early period after the outset of the fever, and the result it produced almost immediately on the movement of the population. So early as the 7th of August, at the recommendation of the College of Phy-

¹ Currie, pp. 79, 144; Condie and Folwell, p. 75.

² Additional Facts, p. 33.

³ Condie and Folwell, pp. 105, 6, 7, 8.

⁴ Condie and Folwell, p. 55.

Currie gives a different enumeration: from 1st of August to 3d of November, 3,446.

August	623
September	1,831
October	942
November	50
							<hr/> 3,446

He adds 60 after the 3d, making 3,506; and 300 in the county. (*Currie*, p. 129.)
Mease says 3,637: August, 626. September, 2,004. October, 943. November, 64 (p. 37).

sicians, the Board of Health suggested to the inhabitants of the infected district the necessity of removing into more healthy parts. The Academy of Medicine, in an official communication published on the 8th, recommended the same measure, as also that means should be taken to prevent the contaminated parts from being visited by the citizens; that all ships, and putrid articles of commerce, should be removed from the wharves and stores of the city; that the docks, wharves, yards, and cellars, should be cleansed; that the gutters should be washed daily; that a sufficient number of physicians should be appointed to take care of such of the poor as might be affected with the fever; that the citizens should be advised to avoid all the usual exciting causes of fever—intemperance, fatigue, excessive heat, the night air, and all violent and debilitating passions of the mind; and in every case of indisposition, however slight in appearance, to apply immediately for medical aid.¹

These recommendations, though proper and advisable, called, in a forcible manner, the attention of the public to the existing danger. Terror soon seized upon all classes. The alarm became general, and emigration was carried to an extent far beyond what had occurred in preceding times of like calamity. The public offices and the banks were removed to the adjoining villages, and even physicians, few in number, it is true, announced their intention of leaving the city.² From Condie and Folwell, we learn that the number of the inhabitants who fled was variously estimated at from three-fourths to five-sixths of the population. They feel disposed to view the first as probably the correct estimate, and lay down at 40,000 the number who sought refuge in the purer air of the neighbouring towns, or of the country (*Ib.*, p. 55); leaving a comparatively small number exposed to the infection and to share the loss occasioned by it.

Some idea may be formed of the malignancy of the fever from the fact of the immense proportion of deaths to the number of persons attacked. The number of cases reported to the Board of Health amounted to 4,718, including 898 received at the City Hospital. Now, if we bear in mind the deaths reported fell but little short of 3,700 (3,645), we arrive at results of an almost unprecedented character. The proportion of deaths to the attacks was as 1 to about 1.27. That of the mortality in the City Hospital was as 1 to 1.75—the cases being 898, and the deaths 514; while in the city and liberties, where the number of cases amounted to 3,820, and the deaths to 3,131, we have a proportion of 1 in 1.23. These reports commenced only on the 18th, and the admissions at the hospital on the 7th. The deaths, however, till the 7th were 53. We may suppose that, from the commencement of the epidemic to the 18th, there may have occurred 150, which, when added to 4,718, gives us 4,868, or 1 death to 1.34.

The mortality in the early part of the epidemic was less severe than it was subsequently; for, while the proportion of deaths to cases throughout was as 1 to 1.297, that from the 9th of August to the 19th of September was as

¹ Condie and Folwell, pp. 47, 48.

² Condie and Folwell, p. 57.

1 to 1.34—the cases reported being 1,937 and the deaths 1,424. In the hospital, the cases amounted to 535, and the deaths to 276—giving a proportion of 1 in 1.2, or thereabout, instead of 1 in 1.75.¹

WHOLE NUMBER OF CASES REPORTED TO THE BOARD OF HEALTH, AND THE RESULT THEREOF.

	Cases reported.	Deaths.	Proportion.
August	827	626	1 in 1.32
Adding	150		1 in 1.56
	<hr/> 977		
September	2,969	2,004	1 in 1.43
October and November (from 1st to 5th no admissions)	922	1,015	1 in 0.908
	<hr/> 4,718	<hr/> 3,645	<hr/> 1 in 1.297
Adding the 150	150		
	<hr/> 4,868		1 in 1.34

CITY HOSPITAL.

	Cases reported.	Deaths.	Proportion.
August	222	112	1 in 1.2
September	483	276	1 in 1.75
October	193	126	1 in 1.53
	<hr/> 898	<hr/> 514	<hr/> 1 in 1.75

The great mortality of this season, in proportion to the remaining population, finds a ready explanation, doubtless, in the greater diffusion of the poison, but, also, in the fact that all, or nearly all, the inhabitants in easy and comfortable circumstances had fled from the infected city, and that the subjects on whom the disease bore were mostly to be found among the lower classes, who, residing in confined, ill-ventilated, and filthy courts and alleys, and unable to seek the benefit of a purer atmosphere, were more exposed to the influence of the morbid cause. The emigration in 1793 did not exceed, as we have seen, one-third of the inhabitants. Hence, among those who remained, a larger portion were of the better classes, who are less prone than the others to be attacked with the disease, and, when so attacked, have it in a lighter form; whereas, in 1798, few of that privileged class felt disposed to confront the danger. As regards the greater mortality in proportion to the number attacked, it was due solely to the greater malignancy of the disease, which, owing to this circumstance, combined with the unfavourable conditions of the classes attacked—arising from the causes just mentioned, as well as bad nursing, &c.—baffled all the efforts of art, and occasioned nearly as many victims as it did sufferers. Dr. Rush, in taking notice of this disproportion of deaths to the number attacked, attributes it to another cause. It “was owing,” he says, “to the liberal and general use of the lancet in 1793, and to the publications in 1797 having excited general fears and prejudices against it in 1798. Such,” he continues, “was the influence of these publications, that many persons who had recovered from this fever in the two former years, by the use of depleting remedies, deserted the physi-

¹ Currie, pp. 84, 85; Condie and Folwell, Appendix, p. xvi.

cians who had prescribed them, and put themselves under the care of physicians of opposite modes of practice. Most of them died."

The sufferings to which the poor and destitute were exposed during this public calamity, from the apprehension entertained of contagion, the wide diffusion and fatal tendency of the disease, and the panic that had seized upon every class of the community, attained an extent unparalleled in the annals of this country. Trade was completely paralyzed, intercourse was almost suspended, the working people were without employment; and while their dearest affections were torn asunder by the constant inroads of the disease, starvation threatened to fill the bitter cup of their sufferings. The public prints of the day, as also the work of Condie and Folwell—the faithful chroniclers of the epidemic—contain details calculated to make every human heart bleed. It is gratifying to discover in the history of the time, that those who fled from the city were more hospitably and humanely received and treated in places where they had sought shelter than had been the case formerly.¹ Experience had already shown, that no fear of contagious communication was to be apprehended from the effect of emigration.

By some, the early cases were attributed to a contagious virus, which had remained dormant from the preceding autumn in the beds and bedding of individuals who had died of the disease. The greater number attributed, as usual, the fever to importation from the West Indies, and vessels were confidently pointed out—the ship *Deborah*, Capt. Yard, from *Jeremie*; the schooner *Aurora*, from *Cape Nicholas Mole*; the *Ariel*, from the same; the brig *Mary*, from *Kingston, Jamaica*, &c. Faithful to the views he had heretofore entertained and expressed relative to the origin of the fever, Dr. Rush rejected the idea of importation, and referred the calamity to the operation of local sources of infection. Many other physicians—and the Academy of Medicine as a body—adopted the same opinion, which was destined to become soon that of the large majority. While such was the sentiment of these in relation to the origin of the fever, the greater number among them, and all the advocates of importation, adhered to the doctrine of contagion, and affirmed that, however the disease might originate, it was communicated from the sick to the well. But the events of the preceding year had induced some to modify their views on the subject, to limit the possibility of contagion within very narrow bounds, or even to deny it altogether. Those of the present sickly season made new converts. Not a few traitors were now to be found in the camp; and it is from this period we must date the revolution in Dr. Rush's mind relative to the subject, which he frankly avowed a few years after.

1799.—The city of Philadelphia was not destined to enjoy a long respite from the ravages of the scourge by which her inhabitants had been so sorely afflicted in 1798. The fever reappeared, as it did also at Newburyport, New York, and Charleston, the following year, but with a less disposition to spread, and in a mitigated form.

¹ Condie and Folwell, p. 55.

The winter preeeding was cold, and continued so throughout, with the exception of a spell of moderate weather which occurred about the middle of the month of January. During the latter part of February, the whole of March, and the first half of April, the cold was very severe; while the spring months, from April to near the middle of June, besides being wet, were eharacterized by a lower temperature than is usual at that season. On the 12th of the latter month, however, a sudden and considerable echange took plaee in that respeat; the weather becoming at once extremely warm, and continuing so (with a short respite of two or three days) till the 26th. On the 24th, the thermometer (F.) rose to 92° . In July, the heat was less oppressive than in ordinary years, and moderated by the not unfrequent oceurrenee of refreshing breezes. The thermometer seldom rose higher than 82° . Much variableness of temperature was experienced in the eourse of August, the thermometer rising on some days as high as 86° ; at others, falling as low as 76° . The same eharacter of weather prevailed in September, regard being paid to the more advaneed period of the season. The alternations of hot and eold days were frequent and sudden. In the early part of Oetober, there was a spell, of several days' duration, of very hot and ealm weather; but, on the 17th, the thermometer fell suffieiently low to produee a severe frost, followed by a eonsiderable and benefieial echange in the atmosphere. But, though marked by variableness of temperature, and tardiness in the appearanee of the spring, the season was suffieiently warm to furnish no less than forty-five days in which the thermometer rose to 80° .¹

June was eharacterized by mueh dryness; no rain fell from the 12th to the 26th. In July, there was some falling weather; and several of the ordinary thunderstorms of the season, aeompanied with heavy showers, oceurred. The month of August was mueh wetter than the preeeding had been; while, in September, rain fell to a eonsiderable amount.

The disease this year broke out somewhat earlier than it had heretofore done, a eircumstanece which, judging from the eharacter of the temperature and weather, might have been expected. It had been preeeded, in the latter part of the winter, by what we have long denominated in this eountry bilious pleurisies; and in April, the diseases were, in general, bilious and inflammatory; while, in the month of June, bilious remittent fevers, some of which were eharacterized by symptoms of great severity and malignaney, were frequent in various parts of the eity. On the 28th of that month, June, the College of Physieians informed the Board of Health that a malignant fever of the same nature as that which had oceurred in Philadelphia in the years 1793-7-8, had appeared in Penn Street and its vicinity. It reeommended, at the same time, "the removal of all the vessels, as well as the inhabitants, from the neighbourhood, and the prevention of intereourse between the inhabitants of the infeeted part and those that were still healthy."

On the 2d of July, the College reeeived an answer from the Board of Health, informing them that they could not eoineide in sentiment with them regarding

¹ Caldwell, Fever of 1805, p. 17.

the propriety of issuing a proclamation enjoining directions for the removal of the inhabitants from the part of the town they had specified, or the vessels from the wharves adjoining; because such public notification would, perhaps, excite a terror that might add to the predisposing cause of the disease, if any such existed. The Board was convinced of the necessity of early precaution, but it also "dreaded to give an alarm which must injuriously affect the welfare of the city, and might, perhaps, eventually be unnecessary; the consequence of which, as it regarded the health, was doubtful, but which would certainly operate against the interest of the citizens."¹

The fever, thus early established, spread but slowly during the course of July, and, gradually diminishing towards the beginning of August, almost disappeared during the first two weeks of that month. Soon after this, however, it revived, spread extensively and rapidly, and continued so to do until arrested by a thick frost in October. On the 22d of August, its existence was publicly announced by the Board of Health, and measures were taken to diminish its ravages. Penetrating to almost every part of the city east of Seventh Street, beyond which very few cases occurred, it principally affected the wharves between Pine and Lombard Streets, near the southern boundaries of the city, and the District of Southwark, in the vicinity of the Swedes' Church. (*Currie*, p. 5.) In this season, the village of Kensington, which in former epidemics had usually suffered, was spared.

The disease, though of long duration and spreading over a considerable surface of the city and districts, did not occasion as large a mortality as might have been feared or anticipated. The whole number of deaths which occurred, from the 21st of August to the 18th of October, amounted to 720, including those reported from the City Hospital; while it is supposed that the average number of deaths in the city and liberties, from the 21st of June to the 21st of August, did not exceed 10 a day, including yellow fever cases; a proportion smaller than that experienced during many seasons free from the disease. (*Currie*, p. 26.) And, again, the return made by the Board of Health to the Governor shows that the interments, from the 10th of July to the close of October, did not exceed 1,276. (*Ib.*) If, bearing in mind the number of deaths reported from the 21st of August to the 18th of October, and recollecting, at the same time, that the mortality from the 21st of June to the 21st of August was about 600, all diseases included,² we endeavour to form from these several data an estimate of the whole number of deaths from the fever, from the 28th of June to the close of the epidemic, we shall, I think, be safe in placing the amount at about 1,000. Dr. Mease³ gives it at 1,015, distributed as follows: August, 375; September, 465; October, 175.

As to the number of individuals who were attacked by the disease, and the proportion of these to the mortality, nothing positive can be ascertained. All I find is, that, during the epidemic, the admissions into the City Hospital amounted to 324, and the deaths to 193; being in the proportion of 1 to

¹ *Currie*, p. 10.

² *Currie*, pp. 25-6.

³ *Picture of Philadelphia*, pp. 37-8.

1.67, or more than one-half. At the Lazaretto, or Marine Hospital, 95 cases were admitted; of these, 21 died.¹

The emigration this year was very great, and the citizens did not wait till the Board of Health had officially announced the existence of the disease. So far, indeed, from such being the case, contrary to the declared opinion of the Board that there was not sufficient ground for the great alarm which pervaded the city, terror spread rapidly. The inhabitants fled in every direction and from every quarter; so that, by the first of September, a great part of the city, as well as of the district of Southwark, was almost depopulated.

The limited mortality from the epidemic, compared to the loss experienced in former years, depended, doubtless, on a less degree of diffusion of the cause of infection and of malignancy in the disease when once excited into action; but it must also be attributed, in a great measure, if not principally, to the considerable emigration which took place. Dr. Rush informs us that soon after the announcement of the epidemic, a publication from the Academy of Medicine, in which they declared the seeds of the disease to spread from the atmosphere only, produced a sudden flight of the inhabitants (iv. 257). In no year since was the desertion of the city so general.

The College of Physicians, faithful to the theory so long entertained by it in relation to the causes of the disease, assigned to the epidemic of this year, as it had done to those of preceding seasons, a foreign origin. It alleged, in support of this opinion, that the fever had been imported in former years, and, therefore, could not owe its development on this occasion to any other cause. Vessels were discovered that had been the instruments of transmission of the seeds of the disease from the West India Islands to the ill-fated city. So strong, indeed, was the conviction in the minds of the majority of both citizens and physicians as to the correctness of the views thus strenuously upheld by the College, that they would not for some time, knowing, as they did, that the quarantine laws had been faithfully executed, admit the truth of the early reports relative to the existence of the disease. It was not until a vessel was discovered, that had arrived from one of the West India Islands on the 14th of May—one day before the quarantine law was put into operation—that they would consent to acknowledge the yellow fever to be exercising its effects among them.

But, while the doctrine of importation was thus defended by the College of Physicians, by many medical men not of that body, and by the public generally, the contrary view was not less zealously upheld by the Academy of Medicine collectively, and by some of its members in their individual capacity. Discarding the idea of attributing the disease to an exotic source, the Academy declared, as they had done before, that it was generated in the city from domestic morbid exhalations; alleging, in support of this opinion, that they saw the fever only in the vicinity of such exhalations, and discovered no channel by which it could have been derived from a foreign country. (*Rush*, iv. 259.)

¹ Currie, p. 26.

1802.—During the summer and autumn of the two years succeeding the epidemic just described, the yellow fever did not manifest itself otherwise than in the sporadic form. In 1800, a few cases presented themselves in the course of July and August, though particularly towards the latter part of September. The greater number of these, not less than twenty-one, appeared in Spruce Street, between Front and Second Streets; of this number 14 died. In the year following, also, a smaller number of cases occurred in July and September, most of which terminated fatally.

In reference to the sickly season of 1801, Dr. Caldwell remarks: "The three summer months had been cool and pleasant, and the falls of rain, though not very profuse, had been sufficient for all the purposes of nature. Corresponding to this constitution of the weather, the diseases of the season had been mild and manageable. Contrary, however, to general expectation, September commenced with uncommon heats, which continued without abatement till the 10th of the month. Throughout the whole period, the thermometer seldom sank during any part of the twenty-four hours below 78° or 80° of Fahr., and it sometimes stood as high as 88°. Not a drop of rain fell, nor scarcely a cloud appeared to intercept for a moment the scorching sunbeams. The consequence of this uninterrupted series of hot and dry weather was, that, about the middle of the month, a pestilential fever broke out near to the drawbridge, and in a short time swept off upwards of twenty of the inhabitants; though it was greatly checked in its course by the cool weather of October, it did not entirely disappear till the beginning of November."¹

In no instance in this year or the preceding, could the disease be traced to a ship, or to a direct or indirect intercourse with persons affected with it. Hence, while by Dr. Rush and others it was maintained that they all occurred in the neighbourhood of putrid exhalations, I cannot find that the advocates of importation made very strenuous efforts to assign to them a foreign origin.

While Philadelphia was thus spared the distresses of an epidemic visitation, other cities of the Union were less fortunate. In 1800, the fever prevailed extensively in New York, Providence, R. I., Norfolk, Baltimore, and Charleston; and every medical reader must recollect the devastation produced by it in Cadiz, Seville, and other cities of Spain; and, in 1801, it broke out in several of the cities of New England, and spread so generally in New York as to produce a considerable desertion of the city. The extended prevalence of the fever in so many places, and its occurrence sporadically in Philadelphia, indicate the continued existence of the epidemic constitution so strikingly manifested in former years, and which now, while favouring in the former the evolution and diffusion of the efficient cause of the disease, and giving rise in some places to the development of an epidemic, could not produce the same effect, except partially in Philadelphia, either from its existing in a much inferior degree of energy, or from not meeting with the concurrence of those local influences connected with weather and localities, which experience has shown to be essential to the wide manifestation of the fever.

¹ Fever of 1805, p. 36.

Very different was the result in the year following, 1802, when the yellow fever once more made its appearance among us in the epidemic form. But, on this as on other occasions, it reigned in a less general manner, and proved a source of much less distress and mortality than it had at several former epochs. I cannot find that anything in the character of the weather or temperature of the spring or early summer months, could have led any one to anticipate a greater development of the disease than had occurred in 1800 and 1801. The month of March, as we learn from Dr. Rush, was wet, cold, and stormy, with the exception of a few pleasant days. Frost was common in April and in May. The weather was so cool as to make fires agreeable to the last, the wind blowing chiefly during the time from the northeast; while, in the month of June, the weather was cold, rainy, and hot in succession; and, during the first two weeks of July, there fell a considerable quantity of rain. As regards the degree of temperature during the summer, June presented, at 3 P. M., a mean of 75.7; July a mean of 78; and August a mean of 78; the result for the three months being 77.2.¹

The fever was preceded by the scarlatina anginosa, which, together with the cynanche trachealis, was the principal disease during the winter months. It reigned almost paramount during May and June, and extended into the succeeding month.

While Philadelphia was thus once more visited by the malignant fever, other cities of the Union suffered in an equal and even greater degree. It prevailed extensively in Baltimore, and especially at Wilmington, Del., where its progress, from the outset to the close, was marked by the utmost severity. It also prevailed in Charleston, while sporadic cases of the disease occurred in New York, Boston, and Portsmouth, N. H. Here it made its appearance, in isolated cases, on or about the 4th of July; the disease, in these instances, being sufficiently well characterized to be recognized as such by Drs. Currie and Cathrall (pp. 6, 7). Its epidemic career did not commence, however, before the 15th of July, when it broke out near the corner of Front and Vine Streets. On the 2d of August, it extended to other parts of the city, particularly Front and Water Streets, near the drawbridge, as well as to Chestnut Street, near the wharf. The disease continued to prevail in different parts of the city during the months of August and September. The number of the cases increased after a white frost. The fever declined after a black frost, which occurred on the 28th of October, and ceased entirely early in November.

As may very readily be imagined, the epidemic diffusion of the yellow fever in Philadelphia was a source of considerable alarm to the inhabitants, who had passed through many severe ordeals of the kind. But this alarm, great as it might have been, was much increased by the course pursued by the Board of Health. On the 4th of August, it publicly announced that the disease had made considerable progress, and that the mortality had been very alarming since its reappearance; though every part of the city and liberties, as far as they could learn, excepting the neighbourhoods adjacent to the place

¹ Cadwallader Evans, Eclectic Rep., vii. p. 428.

where the fever originally appeared, remained remarkably healthy, and entirely exempt from any disease marked with malignant or dangerous symptoms. Dreading, as was stated, the renewal of the melancholy scenes of former years, and finding it would be impracticable, with their limited powers, to prevent intercourse with the infected, they declared the fever to be contagious, and recommended to the citizens to withdraw into the country. The advice, says Dr. Rush, was followed with uncommon degrees of terror and precipitation; and the almost entire population poured into the country, where they remained until publicly invited to return by the Board of Health on the 5th of October.

The mortality from the fever amounted, according to one authority,¹ to 835, and was distributed as follows: August, 262; September, 284; and October, 289. By Drs. Currie and Cathrall, the number is differently estimated. According to these writers, the mortality from all diseases, from July to October, inclusive, amounted to 1,108; *i. e.*, July, 261; August, 262; September, 284; October, 301 (p. 17). The number reported as affected, during the epidemic, was 598; of which number 307 died. During the same period, there died of other diseases 505 persons; making, altogether, 1,069. (*Ib.*, p. 17.) In the hospital, 110 patients were received, and of these 58 died. Admitting these statements to be correct, it follows that the proportion of deaths to cases was as 1 to 1.68. In the hospital, it was as 1 to 2, or fifty per cent. The greatest mortality, in proportion to the number of sick, occurred between the 15th and the 25th of October.

By one party, the fever was asserted to have been the offspring of importation, and was said to be derived from contagion conveyed to Vine Street wharf in the timber of a vessel from one of the West India Islands, and also in the schooner *Eliza*, from Cape Haytien, which arrived near the drawbridge on the 14th of July, laden with hemp, a portion of which was damaged. But, by another party, it was proved that such could not have been the case, inasmuch as the first of these vessels had been detained one and twenty days and well cleansed at the Lazaretto, and that no one of fourteen men who had worked on board of her afterwards had been affected with sickness of any kind. (*Rush.*) The fever by this party was attributed to local causes of exhalation abounding in the various localities which were the seat of infection.

1803.—The year following was another of epidemic.² But, on this occasion, as in others since 1798, the disease was not extensively diffused. It was not on Philadelphia alone that the epidemical constitution of the atmosphere exercised its deadly influence. It spread over different portions of the Union, and, meeting in New York and Alexandria with the usual causes of the disease, favoured its development there in an epidemic form.

The spring of that year was cool. The beginning of May was very much so, and presented the unusual phenomenon of ice so late as the 7th of that month; the wind blowing during the greater part of this and the previous

¹ Mease, *op. cit.*, p. 38.

² See Caldwell, *Med. Rep.*, vii. p. 143. See also same work and volume, p. 187.

month from the northeast. June also was cool. In July, the temperature alternated considerably—being at times hot, at others moderate or cool, with but little rain. But, though such was the character of the weather during the month, heat, accompanied with other unpleasant and hurtful conditions of atmosphere, prevailed. “In July,” says Dr. Caldwell, to whom we are indebted for a brief but clever history of this epidemic, “the cool refreshing northerly breezes of May and June were now exchanged, either for oppressive calms, or for the humid and sultry winds of the south.” The thermometer was seldom under 80° , and sometimes rose to 90° and more. In August, the weather was similar to what it had been in July; but more rainy. During the season, the thermometer rose fifty-one times to 80° and upwards, at 3 o’clock P. M.¹

The mean temperature of June, according to Cadwallader Evans, was 76.9; that of July, 81.3, and that of August 79.4; giving an average for the three months of 79.3.²

The yellow fever was this year preceded and accompanied by remittent and intermittent complaints. In the January and February preceding, catarrhs and bilious pleurisies prevailed—the latter appearing in a tertian type, with pain in the side more sensible every other day. In April, whooping-cough, catarrh, and scarlatina were the ordinary diseases; while in May, June, July, and August, fevers of the intermittent type were, more or less, general.

The malignant fever appeared about the latter part of July. Dr. Caldwell states that two cases (deaths?) occurred on the 19th; and Dr. Rush informs us that between the 25th and 31st, three deaths took place. Other cases occurred in the early part of August. On the 12th, a change of weather put a stop to the fever, and the city remained free from it until the close of the month, when it broke out afresh with more severity than before. In September, the disease spread extensively, and assumed the character of an epidemic; its existence being publicly announced by the Board of Health on the 12th. On the 5th of October, scarcely a case was to be found; and on the 20th, the city received the joyful announcement of the extinction of the disease.

Its first location this season was about Chestnut and Water Streets. It then appeared in Water and Sassafras; and next, in the lower part of the city near South. Except in courts, alleys, and the unventilated dwellings of the poor, its range did not extend to the westward of Second Street. Nor did it, in many parts of the city, ever reach so far. The whole of the city and liberties, except that portion which lies between Second Street and the Delaware, remained as free from disease as it has ever been known in the most healthy seasons.³ The principal focus extended from Market to Walnut Streets, and from the east side of Front to the Delaware.⁴ There the disease assumed its most malignant character. It became milder as it ascended westwardly.

¹ Caldwell, *Fever of 1805*, p. 17.

² *Eclectic Repertory*, vii. p. 428.

³ *Med. Rep.*, vii. p. 188.

⁴ Caldwell, vii. p. 148.

Though characterized by the usual phenomena which serve to distinguish it from other diseases of kindred nature, the yellow fever of 1803 assumed a much milder or less malignant form than it had assumed at most preceding periods. It was less violent in its symptoms; was subdued by less energetic means; and produced a smaller amount of mortality proportionate to the number attacked. Dr. Rush, who particularly notices this fact, states in confirmation that, "in the year 1802, nearly all the persons who were affected with the fever in the neighbourhood of Vine and Water Streets, and in Water between Walnut and Spruce Streets, died. This year, but two died of a great number who were sick in the former, and but one out of twelve who were sick in the latter place." It is possible that our knowledge of the peculiar views of Dr. Rush relative to the unity of disease, and of the fact that the yellow fever did not this year chase away, as on other occasions, other febrile complaints, may diminish somewhat our confidence in his statement of the remarkably low mortality from the fever in the places in question, as we cannot be sure he did not regard in the light of yellow fever diseases of less fatal tendency, and which other physicians would consider as of a different nature. Be this, however, as it may, the fact of a less degree of malignancy in the disease can admit of no doubt. To what cause this must be attributed, whether to a less concentrated state of miasmatic exhalations according to some, or of the contagious poison according to others, or to the co-operation of a less active epidemic constitution of atmosphere, it does not behoove me to inquire here. The subject will be taken up in a subsequent place.

It is not possible to ascertain the number of cases of the fever which occurred during the season in question. From the 15th of September to the 16th of October, 161 cases were reported, besides 27 of a suspicious character. Eighty-seven cases were admitted into the City Hospital. Of these, 49 recovered and 38 died. The whole number of deaths is supposed to have amounted to 195; but on this subject, as well as on the number of persons attacked, no positive information can be obtained. Dr. Rush speaks of the mortality not being more than five in a hundred in the practice of most of the physicians. Supposing this statement to be correct, the deaths having been, as stated, 195, it would result that the number of cases amounted to 3,900; a result impossible to subscribe to. But, as Dr. Rush admits that the cases he alluded to included all the grades and forms of the disease, and as we know that he considered as yellow fever diseases not usually recognized as such, the conclusion is natural, that his estimate of the mortality can afford no clue to the real proportion of cases of the disease which forms the subject of our inquiries.

The doctrine of local origin and non-contagion appears to have made some progress at that time; for the Board of Health, in issuing a declaration of the existence of the disease, affirmed that it was not contagious, and this opinion was entertained and supported by Drs. Rush, Caldwell, and other leading physicians. By others, however, and the public at large, the old and contrary doctrine was maintained with as much pertinacity as ever. The fever was said to have been imported by means of a packet boat from New

York, "because," says Dr. Rush, "a man had sickened and died in the neighbourhood of the wharf where this packet was moored." "It was to no purpose," continues Dr. R., "to oppose to this belief proofs that no sick person, and no goods supposed to be infected, had arrived in this boat, and that no one of these men, who had received the seeds of the disease in New York, had communicated it to any one of the families in Philadelphia, in which they had sickened and died."

EPIDEMICS OF 1805, 1820, AND 1853.

1805.—The year 1804, during which the yellow fever spread extensively at Leghorn, and in not less than twenty-five cities or towns of Spain—Cadiz, Ecija, Carthagená, Malaga, Alicant, Gibraltar, &c.—was one of exemption for Philadelphia and the cities of the United States, with the exception of Charleston and New York, in the former of which it prevailed to a considerable extent. Here, the number of cases was very limited, and in every respect the city was as healthy as in the most favoured years; though remittents and intermittents spread during part of the season more or less extensively in the suburbs; and the smallpox raged as a perfect epidemic.¹ At the same time, the western part of all the Middle, and several of the Southern States, were visited with bilious fevers of all grades and forms.

But while the yellow fever disappeared, with the exceptions mentioned, from the United States during the summer and autumn of 1804, it broke out anew in Philadelphia, as also in New York, New Haven, and Providence, the year after, and, reassuming the epidemic character, spread more extensively in the first-mentioned place than it had done for two seasons before. As in most other epidemic seasons, the summer and autumn of 1805 were characterized in Philadelphia by extreme heat and other atmospheric phenomena already enumerated. The winter preceding had been uncommonly cold and tempestuous. The weather till June was rainy, and not unusually warm. But in this month the summer set in with great severity. On the 14th the heat became intense; it continued so during the rest of the month, and the greater part of July; and scarcely less so in August. For ten days in July the thermometer fluctuated between 90° and 94°; and the register of the weather exhibits an almost continued series of sixty-eight days, in which the mercury at 3 o'clock in the afternoon reached 80°.² The mean temperature of June was, according to Mr. Cadwallader Evans, 75°; that of July, 83°, and of August, 81°. The average of the first two of these months was 79°; and of the three combined, 79.7.³

This intense heat was accompanied by a severe drought, which commenced on the 20th of June, and continued, as Dr. Caldwell informs us, without any intermission, except a few sprinklings of rain that barely moistened the surface of the earth, till the 28th of August. During this period, not only was there no rain, but even the dews ceased to descend. The earth became

¹ Caldwell, 1805, p. 25.

² Caldwell, p. 17.

³ Eclectic Repertory, vii. p. 428.

parched; the pastures were burned up; the supply of summer vegetables failed. Even fruit and forest-trees suffered; and the water in the Schuylkill was lower by several inches than it was remembered to have been for years before (p. 38). Nor did the heats cease with the termination of the drought; and notwithstanding the rain that fell at the close of August and commencement of September, the temperature was not materially reduced, and continued high till near the time of the autumnal equinox. The heat in July and August was particularly oppressive, and suffered no abatement at night; and it is remarked by Dr. Caldwell (p. 39), that even our most cooling winds appeared to have lost their customary effect in lowering the temperature of the atmosphere. The same writer informs us, that the effect of the inclemency of the season was felt by all. While thus oppressed and relaxed by its enervating influence, men "ceased to deem incredible the accounts given of the debility and indolence of the inhabitants of certain tropical countries, whose heats are always intense, and whose soil yields, without cultivation, the means of subsistence." During this extraordinary continuance of heat and drought, the appetite—particularly for solid food—became impaired; thirst became excessive; and the discharge of perspirable matter preternaturally profuse. Bodily strength was necessarily more or less diminished, and with it the power of the system for resisting the action of morbid causes (p. 41).

The outbreak of the epidemic was not preceded by anything peculiar in the diseases of the season. These, as usual, consisted of cholera infantum, dysentery, and ordinary malarial fevers. The former prevailed in June and July to an unprecedented extent; while dysentery, though more particularly diarrhœa, took the place of intermittents on the commencement of the extreme heats of June. They were uncommonly rife among children, and truly epidemic.¹ The yellow fever made its appearance, in a few desultory cases, about the middle of July. Three of these cases occurred in Twelfth Street, south of Walnut; two in the district of Southwark; and the remainder in the southern section of the city. From this period till the close of August, little or nothing was heard of the disease; and the agitation in the public mind, which those cases had created, began to subside. Nothing, indeed, seemed to indicate, in a positive manner, that the city was threatened with an epidemic visitation. At that period, however, the fever broke out with considerable severity in the district of Southwark, about midway between the new market and the Swedish Church. It subsequently reached to Front near Spruce Street, and one case occurred in Pine, between Second and Third. But, though a few cases were observed in the city proper, the principal focus of the fever was circumscribed within the limits of Southwark; most of the city cases being in persons who had resided in or visited that district, and there imbibed the seeds of infection. Epidemically, it did not extend westwardly beyond Second Street in the city, and Fourth Street in Southwark. It was principally rife near the water-side, where it assumed its most malignant garb. It prevailed during the whole of the

¹ Op. cit., pp. 43, 44.

month of September, was weakened by the accession of cold in the first week of October, and finally arrested by frost about the close of the month. But although the disease was limited to a comparatively narrow surface, the mortality from it amounted, as it is supposed, on this occasion, to between three and four hundred. The accurate number of deaths, however, as well as that of the persons attacked, is not known, no report having been made to or published by the Board of Health. By Dr. Caldwell, it is believed that the disease attacked a greater number of persons, and was productive of more mortality than in any preceding year, excepting 1793, 1797, and 1798. "It appears," he says, "from a report of the physicians who attended the City Hospital, that between the 27th of August and 31st of October, the institution received into its wards 346 patients, most of whom were labouring under malignant fever. Were a conjecture on such a subject admissible, I would say, that the aggregate number of sufferers from this fever could not have been less than twelve or fifteen hundred" (p. 53).

While the state of the temperature during the summer months, and the paucity of atmospheric humidity during the greater part of that time were such as has been mentioned, the localities, both in the city and suburbs, were in a condition well calculated, when aided by the unseasonableness of the weather, to begot poisonous exhalations. The health authorities of the time, imbued as they were with the opinion of the foreign importation of the disease, and disbelieving the possibility of its being due to local causes or influenced by them, appear to have turned their attention almost exclusively to the means of preventing the introduction of the fever from abroad, and neglected the pressing and obvious duty of domestic cleanliness. "Such," says Dr. Caldwell, "was their fanaticism on the subject of the introduction of disease from abroad, that they would at any time leave the carcass of an animal putrefying in the street, and filling the air with a poison truly pestilential, to go in quest of a sailor sick only of a last night's debauch, or to meet at the health office for the purpose of passing a resolution to prevent the most clean and healthy West India vessel from entering our port."¹ "From these circumstances," the doctor adds, "together with an entire want of purifying rains, our city became extremely foul, and the atmosphere in a high degree contaminated and offensive. The stench arising from the putrid substances lodged in the gutters, sewers, alleys, and docks, was in many places, to persons unaccustomed to it, quite intolerable. It was, perhaps, more offensive—particularly during the latter part of July, and in the month of August—than anything of the kind experienced by our citizens in former years" (p. 42).

Dr. Rush speaks of a large bed of oysters which had putrefied on Catharine Street wharf, and which had emitted a most offensive exhalation throughout the whole neighbourhood for several weeks before the fever made its appearance.² Dr. Caldwell points out in detail the noxious condition of these oysters. He informs us, that they lay in that situation from the last of

¹ Caldwell, p. 42.

² Med. Inq., iv. p. 96.

June till within a few days of the close of July. By computation, they were never less in quantity than one hundred, and sometimes amounted to at least three hundred bushels. "These oysters and shells were exposed for at least four weeks to a series of the hottest weather that had been experienced in Philadelphia for many years. The consequence of such an exposure need scarcely be mentioned. The mass ran into the highest state of putrefaction, and soon emitted a stench that was quite intolerable."¹

In the course of this epidemic, the removals from the city were not general; neither was the clamor about its prevalence as great as had been the case in former years. Most persons left their homes with more than usual reluctance, and returned to them with uncommon eagerness.² "From the commencement of the disease," says the physician just quoted, "a constant intercourse was kept up between the healthy parts of the city and the sickly parts of Southwark, notwithstanding some strange prohibitory measures and threatening proclamations of the Board of Health. The citizens did not entertain the same dread of the disease which they had manifested in preceding years; and the Board had at an early period rendered themselves too odious to some, and too despicable in the eyes of all, to have any influence either by their remonstrances or their mandates" (p. 52). Dr. Rush, however, states that those families who were in competent circumstances fled into the country.

The measures adopted by the Board, and which, as it appears, gave great offence to the public, and met the disapproval of Dr. Caldwell, were, as near as we can gather, the removal of the sick from the infected district, or the placing of sentinels at their doors to prevent communication with their friends and neighbours, as well as the removal of the poor to tents provided for them in the country. These measures were, however, never carried into effect, except so far as regards a few of the first cases. After a short time, "removals, whether of the sick or of the well, were no longer acts of compulsion, but of choice" (p. 49). The City Hospital was opened for the admission of the sick, and soon became filled.

As in all former pestilential seasons, a considerable difference of opinion existed as to the origin and causes of the fever of 1805, as well as to its mode of propagation. The Board of Health, as already mentioned, were governed in all their measures by the opinion of importation from the West Indies, and of contagion, and endeavoured to promulgate that opinion in all their manifests and proclamations. At the same time, the College of Physicians, and some practitioners of eminence, faithful to their old creed on both points, maintained it with zeal and activity in public prints and different publications. In the *Additional Facts and Observations*, issued by the College in 1806, will be found "an account of the rise and progress" of the fever of the period in question, in which the author, Dr. Currie, makes every effort to trace it to the communication of the individuals first attacked with the Lazaretto. After entering into a minute account of the cases, the

¹ Op. cit., p. 60.

² Caldwell, p. 52.

author says: "From the preceding detail, which the subscriber knows to be substantially correct, it appears evident—especially when compared with the manner in which the disease commenced and proceeded in former years—that it was introduced into Southwark, the present year, by the persons who first sickened at Sam. Crisman's, and that these persons received the infection from some vessel at the Lazaretto, which, from trusting to the care and vigilance of the people of the revenue boat, stationed there to prevent smuggling, had not been sufficiently guarded by the inspectors of the health office" (pp. 94, 5).

A little further on, the same author remarks: "It is also evident, from the dates of the subsequent cases, that the disease was propagated in the same manner as diseases universally acknowledged to be contagious are always propagated, and not like a disease derived from noxious effluvia diffused in the atmosphere to numbers in different directions at the same time; nor like a disease occasioned by foul air, proceeding from a particular source, which ceases to spread, and even to exist, in a few days after the source from which the foul air proceeds has been removed or corrected" (p. 95).

But, while such were the views of the Board of Health, of the College of Physicians, of some other practitioners, and of a large portion of the inhabitants, there were not wanting others who as strenuously espoused the contrary opinion as regards both the origin and contagious character of the disease. Dr. Rush stoutly denied that it had been introduced from foreign parts by intercourse with quarantined vessels, and called attention to the fact that "not a single instance occurred of its being communicated by contagion in any of the families in the city, in which persons had sickened and died with it, or through intercourse of individuals with physicians, and other persons exposed to the effluvia of the sick." Nor was he the only one. Dr. Caldwell, in the excellent history of that epidemic, already several times referred to, has entered fully into the subject, and shown that there was no infected or sickly vessel riding quarantine at the Lazaretto, from which the disease could have been derived; that the persons first attacked had not been actually at the Lazaretto; that individuals who subsequently sickened had had no intercourse with those primarily affected; that, on the other hand, those attacked had been so from exposure to the noxious effluvia arising from the putrid oysters and shells, already mentioned; and that in no instance could the disease be discovered to have manifested a contagious character.¹

The disease exhibited the usual pathognomonic symptoms of the yellow fever of preceding years, and of other cities of this country, of Europe, and tropical climates. It was, however, of a less virulent character than it had been formerly, and was of a more tractable nature when met by suitable remedies in the early stages.² As in former seasons, it was more malignant in the commencement of the epidemic than at a later period. From the commencement of October it was accompanied in general with as little danger as, and was much less obstinate than, a common remittent. The malignancy and danger of the disease were also graduated by its distance from the river.

¹ Op. cit., pp. 55, 66.

² Rush, iv. p. 77.

Persons residing about Third and Fourth Streets, although attacked when the epidemic was at its height, were in less danger than those who lived in Front and Water Streets. (*Caldwell*, p. 51.)

For several years subsequent to 1805, the yellow fever disappeared as an epidemic from Philadelphia. It was met with in a few sporadic cases only, during the summer and autumn of 1806, '7, '8, and '9. But these cases were few in number, and though serving to keep up the remembrance of the disease with the medical practitioners of the city, seldom attracted, to any extent, the notice or excited the apprehension of the public. This form of fever was succeeded by another of an opposite character, which, under the name of spotted fever, typhus petechialis, &c., swept over the Union from one end to the other, and reached Philadelphia in the autumn of 1812, where it spread, as is observed by a contemporary writer,¹ a feeling of gloom and dismay, and will long be remembered as having snatched from their useful and honourable careers some of our most respectable citizens—among whom we have to record a name often mentioned in these pages, and which will ever occupy a foremost rank in the annals of our profession, that of the illustrious Dr. Rush. From 1809 the yellow fever became, as it were, a stranger to our city, even in a sporadic shape, and though it prevailed with almost unexampled violence at New Orleans, Natchez, and Charleston in 1817, as it had done six years before in Brooklyn, nothing was heard of it among us until the summer of 1818, when two well-marked cases occurred and ended fatally. Diseases about this period again appeared to assume somewhat of the character they had exhibited during the prevalence of that epidemic constitution of atmosphere, under the influence of which the yellow fever had for a series of years reigned either epidemically or sporadically, and before the accession of the typhoid character. The preceding year the change became still more manifest. The summer diseases, cholera morbus and infantum, bilious and remittent fevers, as well as dysentery, prevailed more extensively than they had done for some years before, and reassuming, in great measure, their pristine character, ushered in the yellow fever, which broke out unexpectedly in 1819, and threatened to spread epidemically. The first case appeared on the 23d of June on the north side of Market Street wharf, where, from that date to the 6th of July, eight cases of the most violent character occurred. The disease then subsided, and there seemed, for awhile, to be fair reasons to hope, that the city would escape any further extension of the calamity during the remainder of the season. But it reappeared on the 29th of August near the southern limits of the city, Swanson Street and Huddle's Alley, where, up to the 4th of October, fifteen cases occurred. Another case appeared on the 23d of September in Front above Walnut Street. The whole number which occurred in those various localities during the season, amounted to twenty-four. The disease, in all these instances, was of the most malignant kind, and proved fatal in twenty cases. On this occasion, Philadelphia was more fortunate than other cities of the United States,

¹ Jackson, *Fever of 1820*, p. 10.

and some of Europe—New York, Boston, Baltimore, New Orleans, Charleston, Mobile, Natchez, Cadiz, Seville, and Xeres; for while the disease with her proved fatal in only twenty cases, all the other cities enumerated suffered more or less severely, and some to an unprecedented degree.

The fever gave rise this year to much less discussion in regard to its origin and mode of propagation than it had done at any preceding time. A few physicians, and perhaps the larger portion of the public, faithful to their former belief, contended for its introduction from abroad, and attempts were made to trace it to some arrival from a West India, or other infected port. On the other hand, the majority of medical observers denied the exotic origin of the fever, and maintained that it had sprung up in the localities where it appeared and prevailed; that it showed itself under circumstances that prevented its being attributed to any but domestic sources of infection; that it had first appeared among individuals who could not have derived it from abroad, or who had not gone aboard or near a contaminated vessel, or approached a person labouring under the disease, and that there was no vessel in port that could, with any show of plausibility, be pointed out as the instrument of introduction, direct or indirect.

1820.—The slight visitation from the yellow fever which Philadelphia had thus, after a repose of fourteen years, experienced in 1819, was only the harbinger of more serious evils which that city was destined soon to suffer. In the summer of 1820, the disease once more broke out with its wonted energy and malignancy—assumed the epidemic garb, and, had it not been for the judicious and energetic means resorted to with a view to arrest its progress, would, in all probability, have occasioned considerable mortality. It prevailed also in New Orleans.

Dr. S. Jackson, who has published an excellent and interesting account of this epidemic, notices in detail the meteorological phenomena of each month from March to October. From him we learn that the spring was very wet and backward. It succeeded to a winter of greater severity than usual, during which the surface of the earth was almost constantly covered with snow. The quantity of rain which fell in the month of March was considerable; so also in May, when the number of wet days, during which the rain fell continuously or in showers, amounted to not less than eighteen. The rain gauge marked in that month 5.04 inches, while the mean temperature was 66.86°. In June, the summer set in with great severity. The heat was considerable during the almost entire month, which exhibited a mean temperature of 78.06 degrees. The quantity of rain that fell during this was much less than that of the preceding month, amounting to only 1.20 inch. The crops, which had been in a great measure checked by the coldness and moisture of the spring, became, under the influence of the dry heat of June, more luxuriant and abundant than they had been for many years. July was a still warmer month than the preceding, and much more humid. The mean temperature rose to 82.01 degrees, and the quantity of rain that fell amounted to 4.92 inches; the thermometer ranging for several days from 88° to 90°. August and September were warm and dry months; the dryness amounting almost

to a drought. During those two months there were but ten days of wet weather—seven in the first, and three in the second—and the quantity of rain that fell was 1.98 and 1.56 inches. The mean temperature of August was 79.08 degrees, and that of September 75 degrees. About the close of September, the weather became suddenly cold, and on the 25th there was frost. October was characterized by a mean temperature of 58.08 degrees, and remarkable for the quantity of rain that fell—the gauge marking not less than 11.37 inches. The number of wet days amounted to nine. Four inches fell on the 3d of the month, and 3.60 from the 15th to the 16th.

The following table, formed from the foregoing statement, will exhibit, in a compendious and more convenient form, the temperature of each month and the hygrometric character of the atmosphere :—

	Mean temp.	Quantity of rain. Inches.
May	66.86	5.04
June	78.06	1.20
July	82.01	4.92
August	79.08	1.98
September	75.	1.56
October	58.08	11.37

It follows from this, that the mean of June, July, and August, the three months largely concerned in the production and diffusion of the yellow fever, was 79.78. When we compare this with the result obtained during the corresponding months of the two seasons most severely visited by the disease, we find that the mean temperature of 1820 fell short by nearly two degrees of that of 1793, when it rose to 81.37; but was within a fraction of that of 1798, when the thermometer showed an average of 79.94.

While the inhabitants of Philadelphia were placed under the influence of the thermometrical and hygrometrical phenomena just enumerated, they were no less exposed to sources of exhalations in those localities which have ever been the seat of the disease under consideration. Dr. Jackson gives a lamentable account of the condition of the wharves and docks, arising both from the peculiarity of their construction and the careless manner in which they were kept. He enlarges on the effects of damaged potatoes, vegetables, and molasses, which were thrown on the former and in the latter, and which also were permitted to remain in some stores in the vicinity, and allowed there to putrefy and give rise to the most nauseous effluvia. He speaks of the filth of the whole neighbourhood, streets, alleys, as likewise of Pegg's Run, a small stream of water described on a previous page, and which, at the period in question, was in a most foul condition.

Prior to the appearance of the disease, hooping-cough had prevailed, but given way to measles. This in its turn had been succeeded by scarlatina, had which continued to prevail until the month of August, and assumed, in some instances, a very malignant character. (*Jackson*, p. 13.) “In the month of May, a fever, of a bilious and remittent character, combined with typhoid symptoms, appeared among the blacks. It continued to spread during the months of June and July; and late in the last mentioned month it obtained its

height, and was seen in its most aggravated forms. It declined through the month of August, and terminated as an epidemic in September.¹ Between four and five hundred persons were affected with it. It attacked occasionally a few whites of the poorer class, but not more than about twenty or thirty on the whole suffered. It was so generally confined to the blacks that it acquired the name of the 'negro fever.'"

The yellow fever made its appearance this year in the last week of July, the first case being reported on the 24th of that month. From that time to the 2d of August, numerous cases occurred. On the 9th, it recommenced its course, and continued to prevail, with short intermissions, until the last of November, when it was arrested by heavy falls of rain and the accession of cold weather.

As regards the localities affected by the fever, we find that it first broke out in Water Street, near Race or Sassafras, and in a range of buildings situate on what is called Hodge's Wharf, near the foot of the street above named. It prevailed there and in the vicinity till the 2d of August, when its course was arrested by measures adopted for that purpose. On or about the 9th of the same month, it appeared about Walnut Street wharf, and continued to spread there and in the alleys adjoining till near the close of the month, when, by the same means that were used near Race Street, its progress was in some degree arrested. It next showed itself, in a few scattering cases, in Water Street between Market and Mulberry, in Front between Walnut and Chestnut, in Letitia Court, running south from Market, between Front and Second, and in Second Street near Shippen (the first street south of the city boundaries). As the disease was declining in the latter situation and in the centre of the city, it suddenly broke out on the 18th of October in the Northern Liberties, two miles distant from Shippen Street, and one from the other infected districts.

In this situation the disease reached westward to between Front and Second; and we have seen, also, that it prevailed in Letitia Court, situate between those two streets. With the exception of these, as also of the cases in Second near Shippen, and a few in Norris's Alley, running from Front to Second, north of Walnut—the disease did not extend to the westward of Front Street—all the cases being traced to the space included between that street and the river, though principally to the wharves, to Water Street, and the adjoining courts and alleys. To the westward of these boundaries the city, throughout its whole extent, continued as healthy as if no deadly epidemic had existed.

The number of cases reported during the course of the season was, when compared with the results of former epidemics—even of some of inferior note—very limited, amounting to only one hundred and twenty-five. Others, doubtless, existed, which did not come to the knowledge of the public authorities—the mild ones particularly, and even many of those which were of a severe character, though ending favourably. To what extent this system of concealment was carried it is impossible to ascertain; but the cases of the kind

¹ See Emerson, Phila. Med. and Phys. Journ., iii. p. 194.

mentioned could not have fallen short of fifty. To the measures adopted by the Board of Health for arresting the progress of the disease, may be very justly ascribed the limited prevalence of the epidemic. Of the number reported, not less than 83 died, and 42 recovered; being in the proportion of nearly two deaths to one recovery. The following table will exhibit at one view the number of cases, and the relative mortality in each locality:—

SITUATIONS.	No. cases.	Died.	Recovered.
Hodge's Wharf and vicinity	13	9	4
Walnut Street wharves, and Water Street, east side, from Tun Alley to Ross's Wharf, about 700 feet	47	25	22
Walnut Street, and west of Water	8	5	3
Front Street, between Walnut and Chestnut, and Norris's Alley	11	9	2
Front Street, below Walnut Street	4	1	3
Water Street, between Arch and Market Streets	2	2	
Letitia Court, and Market Street	4	1	3
Second Street, near Shippen, and Shippen	5	3	2
Duke Street and vicinity	12	11	1
Scattered in various places, and which could not be satis- factorily traced	19	17	2
	<hr/> 125	<hr/> 83	<hr/> 42

The above table exhibits a circumstance well worthy of remark, and which has been noticed in other epidemics: the greater malignancy and fatality in certain localities. Thus, while at Walnut Street wharves and the vicinity nearly one-half of those reported recovered, and in Front below Walnut, and in Letitia Court the recoveries were still more numerous, three-fourths of the cases at Hodge's Wharf and vicinity, and almost all those in the Northern Liberties, ended fatally.

The disease, as it occurred in 1820, differed in nothing from that of preceding times. Its leading symptoms were the same, and the mortality, in proportion to the whole number affected, was nearly as great as had been observed in the most fatal seasons; thus establishing the fact of the great malignancy of the disease. The latter was easily distinguished into different grades, and presented well-marked divisions—differing in point of severity—calling for a difference of treatment, and constituting the basis of a different prognosis. The rapid progress or diffusion of the disease at the outset, and the greater prevalence of the most malignant class of cases throughout its course, plainly indicate that the epidemic would in all probability have assumed a rank among those of a most formidable kind, had not means been taken to arrest its career of devastation, and limit the sphere of its action. Guided by principles very different from those adopted in former epidemics; unrestrained by the fear of disseminating the sickness into the healthy parts of the city by the removal of the inhabitants of the infected districts, the Board of Health, with its intelligent and indefatigable president at its head, Dr. Samuel Jackson, instead of shutting up the sufferers in the place where they sickened, and placing sentinels at their doors to prevent communication with friends and neighbours, and, at the same time, allowing or forcing those

exposed to the infection to remain within its sphere—the Board of Health, I say, as soon as convinced, from the quick recurrence of cases in the vicinity of Race Street, that the place bore evidence of infection, determined not only to remove such of the sick as had not the means to procure proper attention, and whose situation permitted the change, but to clear the affected district of all its inhabitants. Those able to procure places of refuge, either in the country or in healthy parts of the city, were allowed to shift for themselves; others were provided with accommodations in one of the wings of the City Hospital. By these means, most of the houses were cleared of their inmates in a few days; fences were erected to cut off the approaches to Hodge's Wharf and dock, which appeared to be the focus of the disease; and offensive matters were, as far as practicable, removed. These measures had the desired effect, and put a complete stop to the disease in that vicinity. When the fever appeared at the foot of Walnut Street, similar measures were, at the recommendation of a number of physicians, adopted. The inhabitants of the infected district were as speedily and completely removed as circumstances would permit, and barricades were erected. There, too, the plan was successful. The disease was in great measure arrested in its progress, and limited to the very narrow space already mentioned.

The disease, as usual, was attributed to a foreign source. Some affirmed that it was introduced at Hodge's Wharf by a vessel from St. Jago, which had reached Philadelphia after a passage of twenty-six days. Sundry vessels were accused of introducing the fever at Walnut Street wharf, and in other situations; while the cases which occurred in places remote from the influence of the shipping were attributed to intercourse with those first attacked in the infected district.¹ On the other hand, the doctrine of local infection was loudly proclaimed and ably defended by Dr. Jackson and others, and commanded the support of the large majority of the medical profession. Every account of the supposed importation of the disease was minutely examined and thoroughly sifted; every local cause of infection was pointed out; and the result of the investigation was the disproof, in the minds of that party, of the doctrine of importation, and the establishment of the fact, that the causes of the fever were to be sought at home, and in the places where the cases could almost invariably be traced. This view of the subject naturally carries along with it the doctrine of non-contagion. Hence we find, that though by some the disease continued to be regarded as transmissible from individual to individual, the contrary doctrine was upheld by the almost entire body of the profession. Dr. Jackson, whose opportunities of observation, as President of the Board of Health, were ample, and who has warmly espoused this doctrine, does no more than echo the sentiments of the physicians of the day when he remarks, that the fever of 1820, from its prevailing within certain bounds to which all cases could be traced, afforded the most favourable opportunities of testing its communicability from the sick to those in health. “Nearly one-half of the cases of the disease,” he says,

¹ Griffith, Medical Recorder, xiv. 300.

“were scattered in different parts of the city, evidently contracted in some one of the original seats of the infection that have been designated and described. Many of them were under circumstances in the highest degree calculated to aid its propagation by means of contagion, did it exist. They occurred in the persons of the poor, in confined and ill-ventilated apartments, in houses crowded with inhabitants, in some of the filthiest and narrowest lanes, alleys, and courts of the city, in which the negro epidemic had been, or was still prevailing; yet, in conditions thus propitious to its propagation, not a single instance is known of any person attending on, or who had communion with the sick, or their apartment, having taken the disease.”

The appearance of the yellow fever in a formidable form, after so long an interval, awakened once more the dormant apprehensions of the public generally, as well as of the Board of Health and of the city authorities, for the future safety of the city. Public meetings were held, at which the subject was fully discussed and appropriate measures suggested. By private individuals, schemes of improvement—some of great magnitude, and amounting even to the removal of all the buildings situate between the west side of Front Street to the Delaware, from Vine to South Streets—were proposed. The Board of Health, then under the presidency of the able and active physician already named, was not remiss in taking the matter into consideration, with a view to the adoption of suitable amendments in the health laws then in force; and, finally, the city councils appointed a joint committee to “inquire into the facts connected with the appearance and prevalence of malignant disease, during the past summer and autumn, and to report those means they may deem best adapted to prevent its recurrence, or to check its progress.”

In pursuance of the duty assigned to them, the Committee applied to the College of Physicians, the Academy of Medicine, the Board of Health, the Lazaretto physician, the port physician, and others, for information relative to the probable causes of the disease; the peculiar morbid influences discovered in the infected district, which did not exist in other parts of the city; the agency of frost in arresting the progress of the disease, and the means best calculated to prevent a recurrence of the epidemic.¹

Leaving to the Board of Health the duty of devising such modifications in the health laws, both as regards the subject of quarantine, and of domestic nuisances, as required the attention of the legislature, the Committee of council, fully sensible of the absolute necessity of introducing some modification in the ordinances relative to the cleanliness of the city, in order to insure the existence of a pure atmosphere—which, they were aware, was “no less desirable as a means of preventing the spread of malignant fevers, than as a certain alleviation of them”—thought it necessary to recommend the adoption of a few of the most important schemes required for that purpose, and founded on such facts as were communicated, or were known. They suggested:—

¹ Report of the Joint Committee of Councils relative to the Malignant or Pestilential Diseases of the Summer and Autumn of 1820, in the city of Philadelphia. Philad. 1821.

1. That alterations be made in the mode of contracting for cleansing the streets and alleys with a view to a more effectual fulfilment of that object.

2. That all alleys leading into public streets be taken under the public superintendency, if authority from the State can be obtained; that they be paved by the owners, and in default thereof closed at their intersection with the streets.

3. That some mode be adopted for using the hydrant water at the same time when the scavengers are at work.

4. That hewn stone be laid for a bottom, as well as a side, of gutters, in order to give a clear passage for water and filth.

5. That no wharf be hereafter built, unless the dock on either side be so deep as to be covered by water at *low* tide.

6. That docks now made, and which are not covered at low tide, be filled up, or dug deeper, to produce that effect at the head of the dock.

7. That wharves be paved by the owners, and cleansed as the streets are or shall be; and that when paved, they shall be raised above the summit of spring tides.

8. That privies shall be sunk to gravel, or to the depth of feet where there is none.

9. That no tavern license be granted or renewed, in any part of the city, unless the lot has sufficient space outside of the house for a privy.

10. That no tavern license be hereafter granted for any houses, except those now licensed, and ferry houses, from the east side of Front Street to Delaware River, inclusive; a recommendation predicated on the fact that our summer pestilence has generally commenced and raged with greatest violence within those limits, where, from the confined and filthy character of the localities, the exciting causes of fever arising from intemperate drinking would multiply victims to this disease.

11. That inquiry be made respecting the best means of regulating the number of inmates or boarders in one house, when that number is so great, in proportion to the space for accommodation, as to endanger its health, or that of the public.

12. That a committee of three from each council be forthwith appointed, to inquire into the most expedient measures for improvement in that part of the city eastward of Front Street, with a view to the preservation of the general health.¹

¹ The Report says, pp. 4, 5: "One of the chief objects of your Committee was to obtain a statement of facts; and another, to have the benefit of opinions founded on them. It is well known, that the learned societies who first acceded to the invitation, have heretofore differed in their sentiments respecting the origin of malignant fevers with which the cities of the United States have been afflicted; but your Committee are now happy to observe an unusual degree of coincidence as regards the cause, and entire consent respecting the means of prevention. This unity of medical parties consists in their belief that the city may become unhealthy, even to the malignant state, from offensive matters brought in vessels from other parts, and that a deleterious atmosphere may arise among us from accumulated filth about the wharves and streets, from the want of cleanliness in families, and from inadequate ventilation."

1853.—In the long interval of time which elapsed from 1820 to 1853, the yellow fever prevailed more or less extensively on several occasions, not only in the West Indies and on the coast of Africa, but in several cities of this country and Europe—New York, Charleston, New Orleans, Pensacola, Natchez, Norfolk, Wilmington (N. C.), Galveston, Mobile, Woodville, Vicksburg, Grand Gulf, Barcelona, Gibraltar, &c.

But, while such was the nature of the occurrences in the places mentioned during the above interval, Philadelphia remained entirely exempt from the yellow fever in the epidemic garb. If we except one or two straggling cases which occurred in the summer of 1826, we may, indeed, affirm that the disease was completely unknown among us, even in the sporadic form, during the long space of a third of a century. In the summer of 1853, however, during the prevalence of an epidemic constitution of atmosphere which extended over a large expanse of country, and favoured the development and diffusion of the fever in almost all the West India Islands, in Galveston, New Orleans, Mobile, Natchez, Vicksburg, and many other places in our southern and southwestern States, and even invaded the small village of Brandywine (Del.), it broke out unexpectedly in our midst, and, though not spreading very extensively, attacked a sufficient number of individuals to be regarded as having assumed the character of an epidemic.

The summer was characterized by an unusual degree of heat in Philadelphia, as indeed it was from New Hampshire to Savannah in Georgia. So far as this city is concerned, the following table, showing the mean heat of June, July, and August, confirms the above statement. The observations were made both in and out of doors, and the result varies somewhat from those given before—the difference depending on the locality where, and the hour at which they were noted:—

JUNE.		JULY.		AUGUST.	
in.	out.	in.	out.	in.	out.
77.12	83.88	77.58	83.92	78.89	85.07
80.51		80.75		81.98	

Mean of four observations:—

Out-doors at 7 A. M.; 12 M.; 11 P. M.; and in-doors, 12 M.

75.7

77.1

77.4

In regard to the hygrometrical condition of the atmosphere, it may be remarked, that the quantity of rain and melted snow during the entire year, was less by 3.24 inches than it had been the year before, when we had no yellow fever, and amounted to 42.76 inches. The average of the spring months was 3.84; of the summer months, 4.25; and of the autumnal months, 3.42: the amounts in the first two seasons being inferior to those in 1852, and that in the autumn being but $\frac{22}{100}$ larger than in the corresponding months of 1854. While such was the amount of rain and melted snow collected in the above seasons, the dew-point in each of the months, from April to November inclusive, exceeded (except in October) that in the corresponding months of the preceding years. In 1852 and 1853, April gave 37.10—40.30; May,

48.50—49.29; June, 50.90—54.17; July, 55.90—57.58; August, 55.30—59.31; September, 47—59; October, 48—39.91; November, 35.20—42.22.

The disease made its appearance some time in July. The first case reported, and which attracted the attention of the health officers and the public generally, occurred on the 19th of that month. The patient was a young man, 18 years of age, who drove a furniture-car, with which he stood on the upper side of South Street wharf. On the next day, the 20th, five cases occurred—all among individuals residing along the wharves in the vicinity of South Street. From this point, the disease extended north, south, and west, and covered a space measuring some six hundred yards in one direction and two hundred in another, and bounded north by Union Street, south by Queen Street, west by Second Street, and east by the Delaware River. Of course, cases occurred among individuals residing in other parts of the city; but they were mostly traced to exposure to the above-mentioned infected localities. In some instances, however, no communication of the sick with that locality could be made out. This was especially noticed in some individuals who resided in the upper part of the city, full a mile and a half north of South Street, and were attacked towards the close of September.

The condition of the localities where the disease appeared and prevailed was far from being conducive to health, more particularly under the meteorological influences which prevailed throughout the months of June and July. "Not the least mischievous of these causes," says Dr. Jewell,¹ "in the production of an unhealthy atmosphere, was the outlet of the sewer into the dock at South Street ferry, belching forth continually putrid masses of animal and vegetable filth, accumulating around its mouth, and exposed, at low water, to the rays of the sun, exhaling streams of unwholesome and poisonous gases into the surrounding air. Besides this agent, there was a most foul wharf at the upper side of South Street; a filthy avenue between Lombard and South Streets, without any properly constructed surface drainage; numerous damp and confined cellars, subject to an occasional overflow by the ebbing and flowing of the tide-water of the Delaware; and various minor causes, that might properly be added to the above category, fruitful in the production of atmospherical changes injurious to health." Again, the same writer says the whole neighbourhood "may be considered as favourable to the production and nourishment of malarious fevers, in view of its proximity to the river docks, the open sewer at South Street wharf, the damp cellars, filthy alleys, and other local causes of disease under such a long-continued high thermometrical atmosphere as prevailed during the months of July, August, and September." The gutters, alleys, &c., in other places where the disease appears to have originated, were, for the most part, in an impure condition; while the culvert along Pegg's Run empties into the Delaware at Willow Street wharf, not far from which the three above-mentioned cases occurred, is fully exposed at low water, and contains at all times a large deposit of putrefying vegetable and animal remains.

¹ Trans. of College of Physicians, ii. pp. 54, 64, &c.

As usual, the local origin of the fever from the morbid agency of domestic sources of infection was by some denied. The instrumentality of a vessel from Cienfuegos, Cuba (the barque Mandarin), was had recourse to, to account for the development of the disease, which, it was contended, broke out a few days after her arrival in the neighbourhood of the place where she discharged her cargo. According to that view of the subject, the fever must be referred "to the agency of some uncommon and virulent poison diffused through the atmosphere," which poison, "whatever may be the nature of its character, must, in part, be ascribed to a morbid effluvium, generated under the limber planks in the hold of the aforesaid vessel, from the putrescent state of her bilge water. To this it is added, that the noxious emanations which had been latent in the hold, under the limbers of the vessel, had an opportunity, after the cargo was discharged, and when acted upon by certain causes—as heat and moisture—to disseminate themselves; and that, by coming in contact with other elements of decomposition existing on shore and in the docks, they soon poisoned the atmosphere of the immediate neighbourhood to where the vessel lay moored, and thereby created the evils referred to."¹

By others, however, a different view of the subject is entertained. The agency of the vessel in question is denied, and the epidemic of 1853, like that of preceding years, is ascribed to the morbid influence of local causes. Of the reasons assigned in support of this opinion, I shall not say much in this place, reserving the consideration of the matter to a future part of this volume. All I need say is, that the agency of the vessel in producing the effect contended for, is principally denied on the ground that no proof exists of any poison having been generated in any part of the vessel; that, even had such been the case, we have no evidence of such a poison being capable of acting as a ferment by mixing with domestic exhalations—which alone would produce no deleterious effect—and thereby creating a morbid agent, which continues to exercise its baneful sway months after the departure of the culprit vessel; that such domestic exhalations have often, here and elsewhere, alone given rise to the same kind of fever, and may very readily be supposed to have been adequate to the task of producing a like effect at the time in question; and that, besides, facts of an undeniable character show, that the disease had made its appearance some ten days before the arrival of the vessel which is supposed to have brought it, and a fortnight before the first case reported.²

While such is the diversity of opinion entertained respecting the origin of the yellow fever at the point mentioned, it is admitted on all hands, even by those who deny its having been the offspring of domestic causes, that, when once established among us, the poison did not exhibit a contagious character. So far from entertaining such views, Dr. Jewell, the principal advocate of the exotic origin of the disease, remarks: "Upon the first glance at the Mandarin, and the history of her voyage previous to her arrival at Philadelphia, the advocates for a contagious germ for yellow fever, or, in other words, a

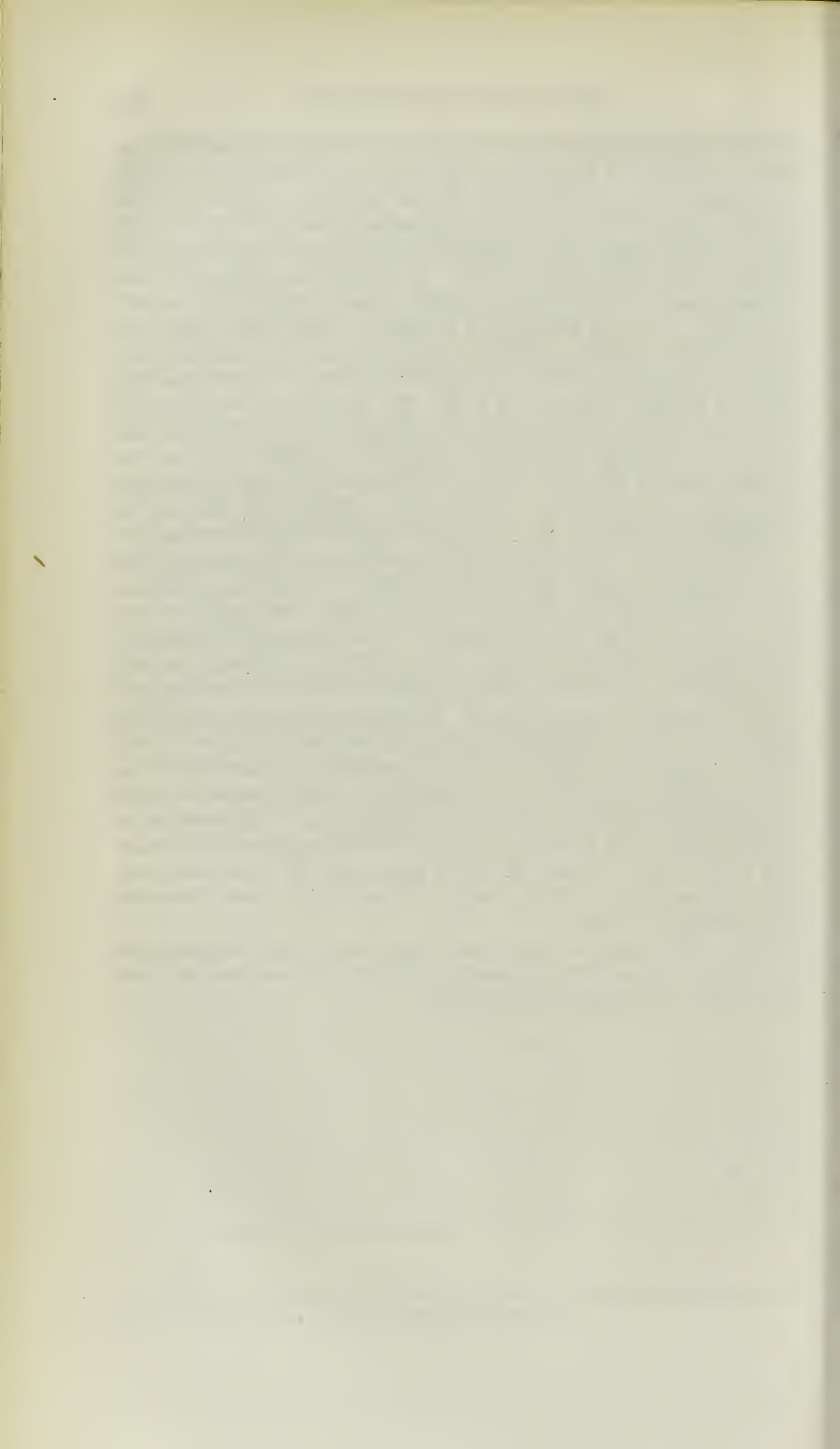
¹ Jewell.

² Remarks on the Origin of the Yellow Fever, which prevailed in Philadelphia in 1853, by R. La Roche, Trans. of Coll. of Phys., ii. p. 214, N. S.

principle emanating from the sick, and capable of being conveyed from one person to another, as the focus for the fever which has threatened our city, may imagine they have discovered another instance in support of their favourite theory. This, however, we are persuaded can hardly be the case, although we are desirous that a careful review of the facts connected with the ill-fated vessel should speak for itself." It is further stated, that in no case was the disease communicated to any person visiting or engaged in attendance on the sick. "At the Pennsylvania Hospital, the yellow fever cases were intermixed in wards with numerous other patients, some ill, and others convalescing from disease; but not an individual, either among the patients, nurses, or visitors, contracted the fever. The like immunity was observed with the cases treated in the Blockley, St. Joseph's, and Bush Hill Hospitals. In private practice, although numerous cases were attended away from the infected portion of the city, we have yet to learn that the disease in a single instance was propagated from the sick to the well, although there was an unrestrained intercourse between the patients and their immediate friends."

From the 19th of July, when the first reported case was attacked, to the 7th of October, the date of the last case—a period of two months and nineteen days—170 were reported; of these 128 died; being in the proportion of 1 in every $1\frac{42}{100}$, or 75 per cent. of the whole. It is well remarked by Dr. Jewell, that there have been, doubtless, cases that presented many of the symptoms of yellow fever in the forming stage, but which, yielding in a few days to a judicious treatment, were not reported. The genuine characters of others were questioned by the attending physicians, and not reported; while some "practitioners, either from indifference, or else questioning the authority of the Board of Health to require reports of malignant cases of disease, or taking the position of its entire uselessness, did not report at all." It is evident, as Dr. Jewell adds, that, had every case that wore the aspect of a malignant type of fever been recorded, the list of cases would have been increased, while the percentage of deaths would have presented a more favourable appearance (pp. 37, 40).

In subsequent portions of this work, I shall dwell in detail on the sources to which this and each of the preceding epidemics have been attributed, and on the mode of transmission of the disease.



YELLOW FEVER.

CHAPTER I.

MEDICAL HISTORY.

SYNONYMES—ALTITUDINAL AND GEOGRAPHICAL RANGES.

THE disease which forms the subject of the present work has received, from the days of Du Tertre to our own times, a variety of denominations, based, according to the views of different writers, on the place whence it was supposed to have been derived; on some one or other of the prominent symptoms by which it is characterized; on its supposed pathological character, the classes of individuals it is most apt to affect, or some other of the peculiarities it presents. It is the *coup de barre* of the old West Indians; the *mal de Siam* of Labat, Desportes, and other early writers; the *malignant fever* of Warren; the *putrid bilious fever* of Hillary; the *febris Indiæ occidentalis* of Mackittrick; the *continua putrida icterodes Caroliniensis* of Macbride; the *elodes icterodes* of Vogel; the *febris maligna biliosa Americanæ flava* of Moultrie; the *febris putrida colliquativa* and *febris ardens æstativa* of Rouppe; the *typhus icterodes* of Sauvages and Cullen; the *typhus tropicus* of others; the *tritæophya Americana* of Sauvages; the *maladie matelotte* of some old writers; the *Kendal fever* of Hughes; the *Barbadoes distemper* of some; the *endemic causus* of Moseley; the *typhus occidental ou ictérique* of Savarésy; the *bilious or yellow fever* of Williams; the *malignant pestilential fever* of Chisholm; the *causos or ardent fever* of Desperrière, Gillespie, &c.; the *fièvre putride continué* of Pugnet; the *bilious remittent yellow fever* of Rush and his school; the *febbre gialla* of the Italians; the *vomito prieto* and *fièvre amarilla* of the Spaniards; the *Bulam fever* of Pym; the *maladie spasmodico lypyrienne des pays chauds* of Chabert; the *hæmagastric pestilence* of Copland; the *fièvre jaune* of most French, and the *yellow fever* of the large majority of English and American writers.

As a general rule, the disease may be regarded as one of low, flat, and level localities, and as appertaining more especially to hot latitudes. Without prejudging here questions connected with the origin and mode of transmission of the fever, the examination of which must be postponed to a subsequent

part of this work, I may remark that it differs, in respect to its altitudinal and geographical limits, from diseases characterized by contagious properties, which this fever is also supposed by some to exhibit, and approximates on the same points to fevers of a local and malarial origin. The former, whether febrile or otherwise, are uninfluenced by any degree of elevation above the level of the sea to which they may happen to be carried. Not so, however, diseases arising from, or connected in some way or another with, malarial exhalations; for they are found to lessen in point of prevalence as we ascend to a considerable distance, and cease to prevail, however propitious the localities may be to the formation of the poison to which they are due, when we reach a certain degree of elevation.

As regards the cause of the diminution and cessation of miasmatic fevers at certain degrees of terrestrial altitude, it will be unnecessary to enter at any length in this place. The effect may be due, in some measure, to a diminished atmospheric pressure. But many circumstances induce the belief that the explanation must be sought principally in the diminished or low temperature which forms a characteristic of high localities; for it is a well-ascertained fact that, except in cases where the soil expands into extensive plains, and the irradiation of caloric which ensues counterbalances the cold incident to such positions, increase of elevation is invariably attended by diminution of temperature. It has been computed that a perpendicular height of from three hundred to four hundred feet produces a decrease of heat equal to that attendant on an approach of one or two degrees towards the poles. Fuster, in his highly interesting work on the *Diseases of France*, calls attention to the fact that, under the line, a degree of cold is generally found to correspond to an elevation of 219 metres, or 730 feet; in the temperate zone, to 174 metres, or 580 feet; in winter, to 70 metres, or 233½ feet less than in summer; and at seven o'clock of the morning, to 60 metres, or 200 feet less than at five o'clock of the afternoon. In Paris, during the hot season, when the ground is nearly as much heated as it is in tropical regions, it was ascertained by Gay-Lussac, at the time of his aerostatic ascension, that at a height of 7,000 metres, or 23,333½ feet, a degree of cold corresponded to an elevation of only 174 metres, or 500 feet.¹

Supposing these statements to be correct, it follows, as M. Boudin remarks, that at the 46th degree of north latitude, an elevation of 2,000 metres, or 6,666⅔ feet, would give us the temperature of Lapland.² With this before us, we can understand that febrile complaints, which are found to diminish in point of frequency as we proceed in a northerly direction, and finally disappear when we reach a certain point, will equally cease to exist at a given height above the level of the sea.

Like the latter class of disease mentioned, the yellow fever never shows itself beyond a certain elevation. It may even be said that, on that score, it manifests a greater difference from the former than ordinary paludal fevers;

¹ Fuster, *Des Maladies de la France dans leurs rapports avec les saisons, &c.*, p. 33.

² *Op. cit.*, p. 35.

for the limits of its altitudinal zone are more restricted than theirs. In the West Indies, we find that Maroon Town and Phœnix Park (Jamaica), each at a height of 2,000 feet, are remarkable for healthiness.¹ Major Tullock, who states the fact, adds, that "while the pestilence of yellow fever rages in the low grounds, and along the coasts of that island, cutting off its thousands annually, their elevated regions enjoy a complete immunity from its effects; for that bane of European life has never been known, in any climate, to extend beyond the height of 2,500 feet."²

The inner Cabrite and the outer Cabrite—the first at 430 and the latter 590 feet of elevation—have also been found very healthy. "In the beginning of the French war, about the year 1756," says Lind, "when the French neutrals were removed from Nova Scotia, a ship, bound to Virginia, on which they were embarked, was driven by stress of weather to the island of Antigua. This mixed company of men, women, and children were all sent to Monk's Hill, in order to recover from the fatigues of their voyage. Soon after this, a general sickness raged in the island. Our seamen in English Harbor, during it, suffered a great mortality by the yellow fever and flood; but the English soldiers, who composed the garrison at Monk's Hill, and the neutral French, though but lately arrived from their cold native country, Nova Scotia, enjoyed a most perfect state of health, and were totally exempted from the prevailing distempers of the island."³ This is usually the case in Antigua, Dominica, St. Jago, &c. In the island of Grenada, Morne Cardigan, 500 feet, and Richmond Heights, 730 feet, are not sickly.⁴ Mount Desmoulin, near Roseau, in the Island of Dominica, at an elevation of 1,500 feet, has been invariably free from yellow fever.⁵ The same immunity has been noticed in St. Domingo, in the mountainous parts of which, whatever be the condition of the soil, this disease does not prevail.⁶ In 1792, when troops freshly arrived from France, and therefore susceptible to the disease, were sent against the revolted negroes, camps were established at the foot and on the summit of the mountains. The fever appeared in the former, but did not show itself at all in the latter. In 1796, the English took possession of the western portions of the island. Military posts were established in the plains and on the summits of the mountain; fever occurred at Port au Prince, in June, but did not reach Mirbalais or Grand Bois. In 1802, similar results were obtained. The same fever has made its appearance, in 1812, and on other occasions, on Brimstone Hill, St. Christopher, at an elevation of 700 feet,⁷ and four times (1817, 1825, 1827, and 1831) at Stony Hill, Jamaica, the height of which is 1,360 feet.⁸ But these are generally healthy, and free from ordinary fever. In Mexico, according to Humboldt, the Farm of the Encero, the height of which is 928 metres (3,243 feet), forms

¹ Imray, Ed. J., lxx. 260; Arnold, 192; Statist. Rept. of Sickness, &c., p. 63.

² Report of British Army, Tullock, p. 43.

³ Hot Climates, p. 223.

⁴ Hunter, p. 307.

⁵ Imray, Edinb. J., lxiv. 340; Lind, p. 224.

⁶ Bally, pp. 326, 335; Dalmas, pp. 64, 65; Gilbert, p. 102.

⁷ R. Jackson, Sketch, i. p. 16.

⁸ Tullock's Rept., p. 59.

the superior limits of the vomito,¹ and the same disease scarcely ever passes beyond the ridge of mountains that separate La Guayra from the valley of Caraccas.²

Major Tullock, while remarking that mere elevation to a height of 600 or 700 feet, does not secure a healthy position, as demonstrated by the instances of Fort St. George, at Tobago, of Morne Fortuné, at St. Lucia, and of Mount Bruce, at Dominica, where, indeed, the results were the reverse of salutary, adds that it is proved beyond a doubt that, "at an elevation of 2,000 or 2,500 feet, they are likely to be wholly exempt from that disease, or to encounter it in so very modified a form, that the mortality from all causes will not, on the average of a series of years, materially exceed that to which an equal number of European troops would be subject in the capital of their native country."³ Bally also gives to the yellow fever an analogous altitudinal limit.⁴

In this country, the yellow fever is never known to prevail in very high situations, whatever may be the condition of the localities. But at what point it ceases to appear or prevail, is still an unsettled question. According to Dr. Drake, along the Mississippi River, one of the most elevated spots at which it has appeared, is the summit of the knobs, on which the upper parts of Vicksburg are built. "The elevation I do not know, but suppose it to be about 350 feet. That of Memphis, however, is estimated at 400 feet, and the disease, as we have seen, has occurred there once." "This," Dr. Drake adds, "is the greatest elevation it has reached in the Mississippi Valley."⁵ But if we admit the fever of Gallipolis to have been of the true icterode character, it will follow that the disease has prevailed in the valley at an elevation of some 600 feet. This is not one-half the elevation of other spots where it has attained and prevailed extensively. We have, in all the instances mentioned, an elevation varying in different places from about 500 to 3,243 feet.

Supposing, therefore, the fever to have really prevailed in the Encero Farm—the limits assigned to it by Humboldt—we may assume this as the altitudinal point, beyond which, whatever be the high temperature and the fever proclivities of the country generally, individuals are secure against an incursion of the disease. They may carry it, but it will never originate, and certainly never be propagated, there.

The inability of the yellow fever to be generated at a high elevation above the level of the sea, depends in part on the greater elasticity and purity of the air, on a diminution of atmospheric pressure, and on a more thorough ventilation. But the main cause is the absence there of the degree of atmospheric heat, which, as we have seen, is indispensably necessary for the elaboration of the morbid agent to which the disease is due. For the same reason, in part, though not exclusively, its geographical limits are restricted

¹ Nouvelle Espagne, p. 771.

² Humboldt, Pers. Nar., iii. 392-5.

³ Op. cit., p. 103.

⁴ Typhus d'Amérique, pp. 325, 6.

⁵ Drake, ii. pp. 188, 9.

within certain bounds in a northern direction ; while in a southern, the same effects are produced, as it would seem, by an excess of heat and a variety of influences of a meteorological and telluric nature. Be the causes, however, what they may, on one point there can be no doubt—that the yellow fever has geographical limits, beyond which it does not appear—and, that, within those very limits there are many places where its usual apparent cause would seem to exist ; but where, nevertheless, it has never shown itself, or has done so very seldom. The West Indian Islands, and part of the coast of South and North America, constitute its proper soil. From Brazil to Charleston in one direction, and from Barbadoes to Tampico in another, the causes of this form of fever are in constant, though unequal force, in regard to different seasons and localities.¹ It prevails often, though not very generally, in some places somewhat more north than Charleston ; visits, occasionally, the Atlantic cities of our Middle States, and has ascended as far as Boston ; while in the Mississippi Valley it has prevailed, as already seen, as high as Memphis, perhaps Gallipolis, or even higher. In an eastern direction, but within the same latitudes, it has extended to Cadiz, Xeres, Carthagen, Malaga, Alicant, Seville, Barcelona, and other cities of the coast and the interior of Spain. It has prevailed several times at Gibraltar, once at Rocheford, once at Lisbon, and once at Leghorn. Hence, we find it embracing a considerable portion of the earth's surface. In its fullest latitudinal extension, it reaches to between the 22d and 23d degrees south of the equator, and, on the other side, to the 42d degree on the Atlantic coast ; to the 35th degree on our western waters, and to the 8.56° on the Pacific. Considered only in reference to its legitimate longitudinal boundaries, it stretches from about the 60th to the 97th degree of west longitude ; while, if we take into account those points in Europe most distant from us, where it has occasionally appeared in an epidemic form, it will be found that its eastern limits may be traced to the 2d degree of longitude east of Greenwich. Its true area includes the Caribbean and other islands called the West Indies, and Bahamas ; the contiguous coast of Colombia and Guatemala and the extensive shores of the Mexican Gulf, sweeping from Cape Catoche on the west to Cape Sable on the east, and running thence along the coast of America to Wilmington (N. C.), Norfolk, Baltimore, Philadelphia, New York, Boston, and intermediate towns ; in some of which places it is an occasional, not annual, or even frequent visitor.

Until recently, the river Amazon, which divides Brazil from Guiana, formed the boundary of the disease south of the equatorial line ; for, although it is said to have prevailed at Olinda from 1687 to 1694,² and to have shown itself as far as Montevideo³ in the beginning of the present century, the latter circumstance is open to some doubt ; while, in Brazil, from the close of the 17th century to the middle of the present, the disease was not observed. Since 1850, it has invaded Rio Janeiro, Bahia, Pernambuco, and other places

¹ Wilson on West Indian Fever, p. 125.

² Ferreyra da Rosa, *op. cit.*

³ Humboldt, *Nouvelle Espagne*, p. 761.

of that country. It is, in greater measure, a stranger to the Pacific, having prevailed but once at Panama,¹ twice at Guayaquil, and once at Callao. It does not appear in the East Indies; the fever described by Wade, Fontane Lind (of Windsor), Johnson, Twining, and other writers appertaining to the class of common bilious remittents. It has never prevailed in China, Cochin-China, Singapore, Siam, Ceylon; it has prevailed only occasionally on the African coast, Senegal, and the Gold Coast, and has but three times, in the space of eighty-six years, showed itself in Cayenne.

Within those limits, it has in some one or more places originated and prevailed to a greater or less extent—occasionally or frequently—either as an endemic or as a mild or wide-spreading epidemic.² Beyond these it never shows itself; and though—whether north or south, east or west—it does not reach the point at which common malarial fevers stop, it approximates to these diseases, so far, especially, as its northern or western extension is concerned, in being circumscribed within certain bounds; for they, too, have their limits. The effect in both instances is due to modifications in the same morbid agencies.³

A distinguished physician of New Orleans, Dr. E. H. Barton, to whom the scientific world is deeply indebted for ample and correct meteorological records and valuable observations relative to the etiology of the yellow fever, has called attention to the fact that within the limits of the zone above pointed out, the disease usually commences its epidemic career in the south, and progresses gradually towards the north. First starting in the West Indies, South America, Mexico, on an average, one year with another, in May, or even earlier, it does not reach New Orleans until some two months later, and, as a general rule, does not break out epidemically in our Middle States and Europe, before the latter part of July or the early days of August. Thus, “commencing at Rio in January, it proceeds, after reaching its acme, gradually north, reaching the northern coast of South America in April and May, and the West Indies and Vera Cruz in May and June; it arrives here (New Orleans) usually the latter part of July, and does not reach its northern limits until some time in August and September.” Dr. Barton adds, “that in this mere historical statement it is not intended to be implied that the yellow fever is imported from the south to the north in this regular gradation, but merely that the physical changes inviting and producing its development become evolved as the season advances.”⁴ Again: as the disease commences usually south of New Orleans two months in advance, “so it retires that much earlier, and being a fever whose ordinary duration is from sixty to ninety days, usually terminates when with us (New Orleans) it

¹ Humboldt, *loc. cit.*

² De la Condamine, *Voyage à l'Equateur*, Paris, 1751, vol. iv. Ulloa, *Voyage Historique de l'Am. Mérid.*, i. p. 149, 1752. Jameson, *Rept. of San. Com. of New Orleans*, 1853, p. 141.

³ Leblond, *Obs. sur la Fièvre jaune*, p. 200.

⁴ Report of Sanitary Commission of New Orleans, 1853, p. 285.

is at its maximum intensity. The same principle will apply with more or less accuracy to the regions north of us."¹

If these be laws, then these laws are subject to numerous and striking exceptions. The yellow fever being a disease of hot weather, requiring a high average temperature during the summer months, and manifesting itself in no climate where the temperature is below that average, it is governed in great measure, in regard to its outbreak, duration, and termination, by that condition of atmosphere. It thence follows, that it usually commences earlier in southern latitudes, inasmuch as the thermometer reaches there the requisite average sooner than in the northern sections of the zone. But, as we proceed, facts will be adduced which show that the disease has not unfrequently appeared later in southern latitudes than it usually appears with us—that as far north as this city it has broken out nearly as soon as it does usually in the West Indies; and that, so far from being always a disease of sixty or ninety days, and terminating in hot latitudes when, not only with us, but even in New Orleans, it is at its maximum, it often not only continues its epidemic career in those latitudes long beyond the limits thus assigned to it, but is even prolonged with but little abatement during one or several years.

CHAPTER II.

CLASSIFICATION OF THE YELLOW FEVER.

THE disease which forms the subject of our present inquiries does not vary either in regard to its pathology or its pathognomonic phenomena, in whatever region or at whatever period in the same place it may show itself, sporadically or in an epidemic form. But, although fundamentally and identically the same in these particulars, under the various circumstances mentioned, the yellow fever, like many other diseases, assumes, in different places, at different periods, and often in the same place during the same season, diversified aspects, or characteristic distinctions—proceeding not from any specific difference of nature, but from various degrees of reaction: from a tendency to depression in the vital force; from the preponderance of certain symptoms, appertaining or not to the disease, and varying according to the greater or fewer number of vital organs involved in the derangement of the body, or from other causes, the whole depending on peculiarity of constitution, temperament, habits, or conditions of health of those attacked, on a difference of intensity in the morbid agent, and on numerous contingencies resulting from the nature of the localities affected, the degree of temperature, humidity, and other phenomena in the surrounding elements. What Dr. R. Jackson has said of the fever of the West Indies applies, therefore, with equal truth to the fever

¹ Report of Sanitary Commission of New Orleans, 1853, p. 284.

everywhere. "The former, though arising from one general remote cause, shows differences of form, and, for the most part, differences of action in a stranger and native, or old resident in the climate; and besides this general and marked difference in the fevers of strangers and natives, there are also differences among individuals of the same class, depending upon original qualities of constitutions, or arising from one or other of the numerous adventitious causes to which men are individually exposed."¹

From this it follows that, in the examination of the disease, we notice not one concatenation of phenomena always invariably the same in every case, and succeeding each other, as the disease progresses towards health or death in regular order, but a variety of groups of symptoms which, though linked together by certain phenomena—which, being pathognomonic of the disease, approximating to each other in several other respects, afford very strong and evidence of their being all members of the same family—are yet sufficiently distinct in their general outline and their mode of progression, to justify their being made the subject of separate consideration. Certain of those groups of symptoms, or varieties of the same disease, prevail more generally in some regions than in others; they are also more frequently encountered in some seasons than in others in the same place; and while in some epidemics several or all the varieties are intermingled among the different individuals attacked, in others, the fever assumes much the same character in the majority of those affected. Such being the case, it is evident that the description of the disease as it affects one individual, or one set of individuals, will not necessarily apply to another case, or group of cases. In some, the fever presents itself with marks of inflammatory action of greater or less intensity; in others, that action is almost or totally absent. In some, the pathognomonic symptoms are combined with an element of malignancy and putridity which imparts a totally different aspect to the disease. In a different set this element is absent, and replaced by one of a nervous character. In some instances, phenomena not characteristic or pathognomonic of the disease, and depending on accidental complications, assume the preponderance, and thereby impart still greater differences in the features of the case.

I scarcely need remark that a general description, which would embrace all the phenomena observed under the diversified circumstances mentioned, would, if alone trusted to, owing to the accumulation of opposite and conflicting details, afford but a slender chance of presenting to the mind a clear and correct picture recognizable at the bedside of the patient; and by subjecting the narrator to the necessity of pointing out, as he proceeds, the endless exceptions to the phenomena described, and the several variations in the arrangement of them, could not but involve the whole subject in obscurity, and thereby greatly impair its utility. To obviate this difficulty, and insure perspicuity in the delineation of the symptoms of the disease, writers on the yellow fever, while, for the most part, admitting its identity under all the circumstances mentioned, have not contented themselves with such a general

¹ Outlines, p. 178.

description, but divided the disease into several varieties or classes, offering under each head a tableau of the principal phenomena, and tracing the progress of these from the outset to the close of the attack. As may be readily presumed, some diversity will be found as to the number of these varieties or classes; for the disease not presenting the same aspect, the same intensity, the same character everywhere and at all seasons, it is easily foreseen that the division which would suit the cases observed by one or more writers, would be inapplicable to those that have fallen under the cognizance of other observers. Nor is it less true that the diversity in question depends, in several cases, on the peculiar views of the writers, several of whom describe, as varieties of the disease, groups of symptoms which others would regard as appertaining to other known complaints, and which are rendered somewhat different by accidental and minor complications. Again, it will be found that the number of these varieties vary in consequence of the difference in the foundations upon which they are based—some writers deriving their classification from the diversified constitutions or temperaments of the persons attacked; others from the degree of violence or malignancy of the disease; others, again, from the inflammatory or congestive character of the cases.

But, whatever be the number of the varieties or classes formed of the disease, or the basis on which the classification proposed may be founded, and however advantageous the adoption of the plan in question may, for the reasons stated, prove to the medical inquirer, it is not to be denied that all the divisions heretofore proposed are, to a certain degree, open to objection, and incapable of obviating all the difficulties incident to the correct portraiture of the disease. Hence, in advocating the propriety of classifying the cases observed under different heads, I am far from supposing that the describer will thereby be always enabled to offer distinct and concise pictures of every form under which the fever presents itself. The most complete classification of the variations into which a disease may be divided is at best arbitrary. The very fact of the diversity of the arrangements proposed affords a proof of this. It is doubtful whether, in the instance before us, more than a few cases can exactly suit the picture presented under different heads, for no two cases resemble each other in every particular; they merge into each other on some point or other, however allied together they may be in their general outline. In other words, most cases, though approximating more or less closely to the description given of one class, present some points of resemblance to other cases described under a different head. The writer, therefore, who selects as his standard one case from among those which, bearing a resemblance to each other, he thinks may be constituted into a separate class, or who draws his description of that class from the aggregate of such cases, will inevitably produce a portrait which cannot exactly be applicable to any one case in particular.

Unsuccessful, however, as the attempt to avoid the difficulties mentioned may prove, such difficulties are so infinitely inferior to those which would accrue from the adherence to a different plan—the cases of each class differing from each other much less than they do from those of other classes—

and are so decidedly counterbalanced by the advantages to which I have already adverted as resulting from the classification of the disease under various heads, as to place the propriety of the latter plan beyond the possibility of doubt, and to justify its adoption.

But, while generally admitting the propriety of this course, writers on the yellow fever have, as already seen, differed, and continue to differ, as regards the number and arrangement of the varieties into which the phenomena should be grouped. In this city, several of our writers—Currie, Cathrall, Deveze, Nassy, Barnwell, Eberle, Dewees, and Wood—have paid but little attention to the subject; their descriptions being, for the most part, made to embrace all cases, though followed, in some instances, by an enumeration of exceptions or variations in the order, character, or complication of the phenomena. By Dr. Rush and Dr. S. Jackson, a somewhat different course was pursued, and an attempt at classification made. The former, in his account of the epidemic of 1793, while abstaining from describing separately each variety, and presenting a picture of the fever, divides the patients affected with the disease into three classes. “The first includes those in whom the stimulus of the miasmata produced coma, languor, sighing, a disposition to syncope, and a weak or slow pulse. The second includes those in whom the miasmata acted with less force, producing great pain in the head, and other parts of the body; delirium, vomiting, heat, thirst, and a quick, tense pulse, with obvious remissions, or intermission of the fever. The third class includes all those persons in whom the miasmata acted so feebly as not to confine them to their beds or houses (iii. 74). In the year 1797, the same writer classed the cases into a greater number of forms or varieties: 1. Cases in which death occurred in four and twenty hours, with convulsions, coma, or apoplexy. 2. Open cases, with full, tense pulse from the beginning to the end, severe pains, &c. 3. Depressed or locked cases, with debility, little or no pain, a depressed and slow pulse, a cool skin, cold hands and feet, and obstructed excretions. 4. Divided or mixed cases, with active pulse until the fourth day, when it became depressed, accompanied with all the other symptoms of the locked state. 5. Cases in which the pulse imparted a perception like that of a soft and shattered quill, accompanied with a disposition to sweat. 6. Walking cases, in which the patients were flushed or pale, had a full or tense pulse, no pain, good appetite, and walked about their rooms or houses as if little indisposed, until a day or two, or a few hours before death. 7. Intermittent cases. 8. Mild remittent cases. 9. Mild cases, in which the patients continued to attend to their usual avocations. 10. Yellow or jaundice cases, with slight indisposition. 11. Chronic cases (iv. 13–15).

Dr. S. Jackson, of this city, in his clever account of the epidemic of 1820, already referred to, after remarking (p. 50) that Drs. Physick and Parrish, two eminent practitioners, had observed, in our various former visitations, that three grades of the disease could be evidently distinguished, differing in the concurrence of their symptoms, states that on the occasion in question the disease was easily distinguishable into different grades, and presented

well-marked divisions. These grades or divisions were, according to him, three in number; and of each he presents an elaborate description. The first grade includes those cases in which the disease manifests its greatest violence, and becomes thereby entitled to the appellation of malignant. The second grade includes cases characterized by high inflammatory action; while the third, though possessing the same features and the same outline of symptoms as the other two, is mild and easily manageable, being, in some cases, no more than an ephemera of one day's continuance, or continuing from three to five days, and being attended with remissions (pp. 50, 60, 61).

Different divisions have been proposed in this and other countries. Dr. Chatard, of Baltimore, makes out three varieties: 1. The inflammatory. 2. The bilious. 3. The nervous.¹ Dr. Jameson, of the same city, divides the disease into six varieties: 1. The synocha, or inflammatory. 2. Synochus—a less inflammatory grade, and holding a middle place between the former and the fourth. 3. The synochoid, presenting a still greater approach to the fourth. 4. The gangrenous. 5. The hectic—a termination of some other form, the consequence of uncured local disease. 6. The occult.²

Dr. Hosack, of New York, recognized two varieties, or, as he denominates them, two species: 1. The simple, or inflammatory yellow fever; and, 2. The malignant, typhoid, or contagious fever.³ Girardin, in like manner, marks out two varieties, the inflammatory or sporadic, and the malignant or epidemic (pp. 32, 54). Berthe and Ameller's division differs in nothing from that proposed by Dr. Chatard (pp. 153-4, 368). Dr. Dufour, of Leghorn, considered the disease under three heads: the ataxic, the adynamic, and the simple, or bilious.⁴ Palloni, of the same city, admits likewise the existence of three varieties: 1. Cases characterized by symptoms of vascular excitement. 2. Cases marked by gastric symptoms. 3. Those exhibiting symptoms of debility.

Dr. Robert Jackson regarded the disease of Andalusia, in 1820, as having presented three varieties: 1. As it occurred in persons in whom the sanguine temperament predominated. 2. As it occurred in the persons of those in whom the lymphatic temperament was most conspicuous. 3. As it appeared to exercise a particular influence on the serous capillaries. The first of these forms is distinguished by the usual symptoms of high vascular action. The second is characterized by "defective energy in the circulation, and consequent imperfect function through the whole system." In the third form, "the primary act seemed to be constrictive. It assumed considerable varieties of appearances at its commencement, and was the most frequently encountered in that season."⁵ In his sketch of the febrile diseases of the West Indies, the same writer speaks of the fever as assuming a greater number of varieties, according as it produces a more decided impression: 1. On the sanguine temperament. 2. The gangrenous temperament. 3. The phlegmatic tem-

¹ Obs. des Sci. Méd. de Marseille, v. 337.

² Med. Recorder, vi. 444.

³ Med. Essays, iii. 424, 425.

⁴ Obs. des Sci. Méd. de Marseille, iv. 50.

⁵ Fever of Spain, pp. 68, 69.

perament. 4. The serous temperament. 5. As it attacks principally the sentient and intellectual system.¹ The first, third, and fourth are similar to the varieties observed in Spain, where, however, the other two forms—the sentient, in which the nervous system is functionally disturbed, and the gangrenous, in which the system at once sinks from deficiency in the power of reaction, oppression of forces from the outset of the attack, and stagnation of blood—occasionally, though not frequently, occurred.² Each of these varieties he subdivided—the first, into mild and aggravated; the second, into mild, aggravated, and periodic; the third, into mild, aggravated, and liquescent cachectic; and the fourth, into the constrictive serous form, and a retrograde, colliquative, or liquescent form, characterized by excess of secretion, particularly of the cutaneous; colliquation and swelling of the whole body.

Another writer of eminence, whose experience in the yellow fever was derived within the tropics, regards the disease as divisible into two species. According to him, “the essential character of the first species of the yellow fever arises from the existence of a state of indirect debility which presents itself under the aspect of an eminently inflammatory diathesis, and from an evident sthenic condition of the system. The character of the second species is based upon the existence of direct debility, which resembles the state of synochus, and approximates to that of nervous fever. Regarding the disease as belonging, under all circumstances, to the nosological genus *typhus*, he applies the name of *typhus icteroide* to the yellow fever characterized by an indirect asthenic diathesis—dividing this into two varieties, the regular and the irregular, each of which again assumes two degrees. The second form of the fever, that by direct debility, he denominates *Western or American typhus*.³

Dr. Ralph states that the fever of Barbadoes, in 1816, appeared under four forms. The first was recognized by symptoms of high inflammation during the period of reaction. In cases embraced in the second form, the disease was marked by a state of general depression at the outset, and deficiency of reaction. The third included the milder and more protracted cases. In the fourth form, the disease was deceitful and insidious—very generally fatal, and characterized by little if any reaction.⁴ Sir William Pym recognized at Gibraltar, in 1828, three forms or varieties of the disease; 1. Cases in which the fever was mild and ephemeral. 2. Severe cases, in which the paroxysm was prolonged to fifty or seventy hours, and terminated quickly, the disease presenting all its characteristic features, and often ending in death. 3. The more malignant and rapid cases.⁵ Dr. T. Smith, in his critical review of Pym,⁶ adopts much the same division, which differs only in slight degree from that adopted by Pouppé Desportes,⁷ Gilbert,⁸ Frost,⁹ and

¹ Vol. i. pp. 60, 86, 102, 138, 144.

² Op. cit., p. 107.

³ Savarésy, p. 266.

⁴ Med.-Chir. Journ. of Edinburgh, ii. 65, 72.

⁵ Edinb. Journ., xxxv. 41.

⁶ Ibid., p. 42.

⁷ Mal. de St. Domingue, i. 176.

⁸ Hist. Méd., &c., p. 72.

⁹ Med. Reposit., xiii. 29.

Pariset,¹ the Reporters on the epidemic of 1839 at New Orleans.² Finally, other writers, Wilson,³ Barton,⁴ Merrill,⁵ Kelly,⁶ Cartwright,⁷ Hogg,⁸ Wallace,⁹ O'Halloran,¹⁰ Catel,¹¹ Davidson,¹² Caillot,¹³ Stevens,¹⁴ Dickson,¹⁵ Evans,¹⁶ Wallace,¹⁷ and Copland,¹⁸ have admitted, in some measure, the classification first proposed by Jackson, *i. e.* two principal varieties; the inflammatory, with reaction more or less free and open, and the congestive, typhoid, malignant, or cold form, attended with feeble, if any reaction, and in which death often occurs without the signs of inflammation having presented themselves, and sometimes, apparently, from the exhaustion of the nervous system. To these varieties, Dr. Barton adds a third, which includes what he denominates the simple cases. The first of these writers, Dr. Wilson, to whom we are indebted for one of the best works on the West Indian fever, subdivides the inflammatory and congestive forms of the disease into three grades; the first into the mild, the violent, and the intense; while the congestive he describes under the heads of slight, aggravated, and apoplectic.

For every purpose of description, the division adopted by Dr. Wilson appears the best; enabling us, as it does, to include with sufficient accuracy the various cases, however dissimilar in appearance, which fall under the cognizance of the physician. In its grand outline, the division of all cases into the inflammatory, or those characterized by well-marked reaction and the congestive, in which this reaction is but faint or totally absent, is founded in nature; and it would require but a slight examination to show, that all the other divisions I have pointed out may easily be resolved into the one in question. The first variety, proposed in 1793 by Dr. Rush, may, without difficulty, find its place under the head of congestive—the other two under that of inflammatory. The second variety observed by him in 1797, included cases of an inflammatory kind; the others were modifications of the congestive. The same remarks are applicable to the classification of Drs. S. Jackson, Jameson, Hosack, Palloni, Dufour, Berthe, Smith, Savarési, Ralph, and others, as every one may see in reading the description they have given of the disease in its various modifications. The varieties described by Dr. R. Jackson, as manifesting themselves in Spain, are, as Dr. Good justly remarks,¹⁹ equivalent to the inflammatory, typhus, or synochus of other writers; and these, again, are equivalent to the inflammatory and congestive forms of another set of authors; while it would not be difficult to show that the other varieties admitted by him in the West Indies, as also admitted by Ralph

¹ F. J. de Barcelone, p. 367.

² Report, p. 162.

³ West India Fever, pp. 7, 8.

⁴ Am. J., xv. 36.

⁵ Med. and Phys. Journ., ix. 243; N. A., ii. 223.

⁶ Am. J., Oct. 1847, p. 377.

⁷ Recorder, ix. 13.

⁸ Western Jr., ii. 417.

⁹ Ed. Jr., xlv. 274–8.

¹⁰ Fev. of Andalusia in 1820, p. 74.

¹¹ Chervin's Report, p. 11.

¹² Repos., viii. p. 248–9.

¹³ Traité de la Fièvre jaune, pp. 20, 21.

¹⁴ Obs. on the Blood, p. 272.

¹⁵ Am. J., ii. 70.

¹⁶ End. Fev. of W. I., p. 307.

¹⁷ Edin. J., xlv. 273.

¹⁸ Dict. of Pract. Med., iii. 301.

¹⁹ Study of Med., ii. 167.

and other writers, may find their place under one or the other of these two heads.

As far as the observations collected during the epidemics which have prevailed in this city extend, it may be stated that the fever has exhibited a character which justifies a division somewhat similar to that proposed by Dr. Wilson. That it is perfectly satisfactory on all points, I will not aver, aware, as I am, that, as already stated, no classification can be perfect—the cases merging into each other in the most provoking manner. But it is sufficiently accurate, and may be made to embrace, under their appropriate heads, most if not all cases that present themselves. Before doing this, however, I shall present a picture, drawn from personal observation and from our best authorities, of the aggregate of the symptoms as they have usually shown themselves in this city. It may be premised, from the slight notices we possess of the epidemic of 1699; from the more detailed, but still imperfect descriptions of those of 1741 and 1762, for which we are indebted to Dr. Kearsley,¹ Dr. Redman,² and Dr. Rush;³ from the more elaborate records we have of the epidemics which prevailed from 1793 to 1820, inclusive, as may be found in the writings, already often cited, of Rush, Currie, Cathrall, Caldwell, Deveze, Nassy, Barnwell, Jackson, &c.; and from the phenomena observed during the sickly seasons of 1853 and 1854; that, while in its various visitations from the earliest periods to the year 1854, the disease presented pathognomonic marks, which establish its identity as to pathological character, and which will be recognizable in the picture I am about to lay before the reader, it at the same time assumed at each visitation the diversified aspects by which the varieties in question are distinguished. But here, as elsewhere, at every repetition of the disease, some of those varieties were more frequently seen than others. At times, one variety, in its several modifications, assumed so decidedly the preponderance as almost to exclude the others, and thereby to impair somewhat the correctness of the description if judged by the occurrences of those epidemics, or if applied to each case individually during the same season; nevertheless, taking it as the representation of the disease in its totality, it retains sufficient accuracy to prove useful; and is applicable to so large a number of cases, generally observed, in some seasons more, in others less, as to afford the means of forming a correct idea of the features and mode of progression of the disease, and to establish its relationship to the fever as it appears elsewhere. Its failure in portraying particular cases of less frequent occurrence, or which present an anomalous aspect, will easily be corrected as we proceed in the details of the several modifications of the different varieties, and afterwards in an examination of the principal symptoms by which the disease is characterized.

¹ Med. Mus., i. 20.

² See *ante*, p. 63.

³ *Ibid*.

CHAPTER III.

SYMPTOMATOLOGY OF THE DISEASE.

IN the following description, the symptoms will be laid before the reader as much as possible in the progressive order in which they present themselves, attention being paid to the different stages into which the attack is divided. That a different course has been pursued by Dr. Rush, Dr. Jackson, and others, in this and other places, I know full well, most of them contenting themselves with pointing out the modifications produced by the disease in the various organs and apparatuses of the economy—the sanguiferous, the respiratory, the cutaneous; the gastro-intestinal; in the liver, lungs, and brain; the nervous system; the senses and appetites; the secretions and excretions; the lymphatic and glandular systems; the skin, the blood, &c. &c.—and the phenomena resulting therefrom. Nor am I less aware that, in the yellow fever, from the protean character of the disease, from the fact that few cases resemble each other exactly, and that in the mild as well as some of the worst and most malignant forms, the cases present but one stage—being characterized throughout by symptoms of excitement, or those of prostration—it may sometimes be difficult, if not impossible, to trace with accuracy the line of demarcation between the various stages, which, indeed, are hardly manifest. Nevertheless, an extended survey of the disease, in its several varieties, must lead us to admit, with Deveze (p. 28), that the large majority of cases exhibit several stages in a manner too evident to be mistaken. These stages, according to the same author, are, like those of other complaints, three in number; the first, that of reaction, being in general well marked and defined; as is also its passage to the second, or that of exhaustion; while the third consists simply in the aggravation or maximum of the phenomena constituting the second. (*Deveze*, p. 25.) Nor are the stages of equal duration in all cases; the first continuing from one to three days, rarely beyond; while the duration of the second, as well as of the third, varies very greatly. Other writers, as Copland, admit also the existence of three stages; but, with them, the first stage is that of the cold period; the second is that of reaction; and the third, that of exhaustion (iii. 142). Others, again, make but two stages—that of excitement, and that of collapse, or exhaustion. In the following description, the disease will be considered as presenting three stages: 1, that of reaction; 2, that of calm or remission; and 3, that of exhaustion.

SYMPTOMS.—The attack of yellow fever often commences abruptly in the midst of ordinary health; the patient being seized, without warning, with languor and a sense of debility, which sometimes supervenes while he is walking in the street or attending to his usual avocations. Sometimes it is

preceded for several days by anorexia; the individual complaining also of general uneasiness, and labouring under costiveness, loss of appetite, flatulence, heat in the stomach, giddiness, pain in the head, combined with dull, watery, or brilliant yellow or red eyes, low spirits, &c. In some of these cases, as well as in those in which the disease comes on abruptly, the attack commences sometimes at night—generally in the after or forepart of the day. In some instances, the patient is struck down instantaneously, as if by a blow or by lightning, and falls at once into a state of coma. Very frequently the attack is ushered in by a regular chill, sometimes by a mere sense of coldness; in others, again, by complete rigors; whilst, on the contrary, very severe and dangerous cases are not unfrequently characterized by an absence of all symptoms of the kind.

In those cases in which the attack is ushered in by a chilly fit, the latter often alternates with glowing flushes of heat. This state is generally of short duration; but at times it continues for twelve or more hours. It is usually followed by confirmed fever, which is more violent towards evening and throughout the night, and presents some alleviation—but seldom a decided remission—towards morning. In a different set of cases, on the contrary, and those the most malignant and dangerous, there is an almost or total absence of febrile reaction; the pulse is feeble, soft, sometimes full, or scarcely to be felt, the patient passing at once into the state of depression—presently to be described—or sinking immediately with stupor, coma, and convulsions.

After the febrile excitement has become fairly established, the pulse is generally found quick and tense, and during the exacerbation full and strong, though sometimes soft, beating from ninety to one hundred and twenty times or more in a minute. In very malignant cases, it is gaseous. The temporal and carotid arteries, during this time, throb and beat strongly. The skin becomes hot, dry, harsh, and pungent. In others, it is dry, unctuous or perspiring, flabby and cold, except over the centre of the body. The face is highly flushed, or pale, or purplish; and the eyes remarkably red, sometimes apparently bloodshot, hot, more or less painful, and imparting a sensation similar to that produced by the introduction of grit or sand. In many instances, the ball of the eye appears to be a mass of vessels filled with blood; it is at the same time brilliant, shining, and watery, and in some instances presents somewhat the expression peculiar to drunkenness. In some cases, this state continues from the commencement of the attack to the close of the stage of reaction; in others, it does so only partially for the first and second day. In some instances, there occurs profuse perspiration, which continues to the second or third day; while in others, there is but little change in the temperature of the surface; in others, again, the surface soon becomes dry and cool, with a complete torpor of the vessels, and loss of irritability.

These symptoms are usually, if not always, accompanied with an annoying, and even torturing pain in the head—most commonly in the forepart, and in the eyes, and shooting from temple to temple, but sometimes only on one side. In most cases, the pain continues throughout the whole stage of reaction, and constitutes one of the most distressing features of the disease. In

addition to this, the patient complains in equal degree of pain in the back, loins, and large joints—extending from the loins to the hips, and down the thighs, and even lower. This pain is generally so severe as to extort groans and even screams from the patient. But frequent and severe as these symptoms are, cases occur in which the patient, in great measure, or even altogether, remains free from them; or, in some very malignant cases, the pain in the back and limbs is obscure and dull, or is replaced by a sense of weight and stupefaction in the head. The tongue, during this time, is moist, and covered with a white, thin, cottony fur, and usually, though not always, red on the edges and at the apex; while the throat is occasionally sore, to such an extent, indeed, as not unfrequently to cause difficulty of deglutition.

In almost every case, the gastric organs become early involved in the morbid derangement, as manifested by a variety of phenomena. Nausea, or other uneasiness of the stomach, with or without vomiting, is not unfrequently an attendant from the outset of the attack; the stomach is generally distended, often, though not always, painful on pressure, and irritable, especially after taking any kind of drink or aliment; frequently affected with sick qualms and more or less propensity to yield its contents. But although these gastric symptoms often occur at an early period, they are not generally fully developed until from twelve to twenty-four hours after the outset of the attack, or at the accession of the second stage, when they become prominent. When this is the case, the patient complains of a burning pain, or a sense of stricture, weight, distension, or oppression—sometimes overwhelming—at the præcordia, which feels as if tightly bound with a cord. Tenderness or pain on pressure, if it did not exist before, is now experienced—at least in most cases—and becomes excessive. The irritability of the stomach increases, and proves distressing—the organ rejecting everything swallowed, and throwing off its morbid contents when undisturbed by drink or medicine—the act of ejection being often violent, and attended with retching and considerable distress and pain. During the stage of reaction, the matter thrown up consists usually of the drinks or other substances swallowed—sometimes mixed with clear, glazy mucus, or, in some cases, with matter of a sea-green colour and bitter taste. In mild cases, bilious vomiting sometimes occurs. In those of a concentrated character, bile is seldom ejected, and when it is, cannot be viewed as characteristic of the disease. While the patient suffers in this way, he usually experiences considerable, though not often insatiable, thirst. But most generally his desire for cold drinks is extreme, the natural result of the gastric heat under which he labours. The urine, during this time, is generally deficient in quantity, and of a dark-red colour, often accompanied with a copious sediment.

The bowels are ordinarily costive—occasionally obstinately so. When stools are procured by art or artificial means, the first discharges are generally soft, feculent; sometimes, but seldom, tinged with bile; at others, of a drab colour. When no cathartics are used, the stools in the course of the disease become of lighter colour, and assume a starchy, cream-like or puru-

loid appearance. In a few instances, they are watery, or even bloody, from the outset of the attack.

In the meanwhile, the patient is a prey to restlessness and jactitation; moans, sighs, and shifts his position constantly in search of ease. Even in those cases in which jactitation is absent—and these are not seldom encountered—the patient sometimes feels a disposition to rise from his bed and walk about the room, his muscular strength remaining unimpaired to a degree unusual in febrile diseases. But, in a large number of instances, there is from the outset universal debility, which continues to the last. Respiration in some is laborious and hurried; in others it is slow, and accompanied with deep and heavy sighing. Instances are not unfrequently found in which the patient complains of feeling as if he could not expand his chest and inflate his lungs; or experiences spasmodic pains about the chest. Nevertheless, none of these symptoms are constant attendants on the disease, which not seldom runs its course without greatly disturbing the respiratory function.

When drawn soon after the onset of the attack, and when symptoms of reaction are well marked, and especially when local inflammation exists, the blood is sometimes found of a bright arterial colour, as well as sizzly, and even cupped, as in ordinary phlegmasiæ. In very many cases, if not generally, however, it presents no cup or buffy coat, whilst the coagulum is flabby and easily torn. In some cases, when the separation into crassamentum and serum takes place, the latter is of a natural colour. In others, it is of a yellow hue or slightly tinged with red, and transparent. In another set of cases—and in some epidemics more frequently than in others—the separation does not take place, the fluid remaining for hours, or altogether, much as when received in the vessel. In a few cases, when drawn later, or at all periods in cases unattended with reaction, the blood is of a dark colour, void of all inflammatory manifestations, and not unfrequently as fluid as molasses; while in other instances, again, it is smeared over with a pellicle of sizzly lymph, at the same time that the part lying at the bottom of the recipient vessel is dissolved. In the early stage of the inflammatory variety it is very hot, and has a peculiar odour, which, according to some accurate observers, is supposed to furnish a sure indication of the true nature of the disease.

In the early stage, the mind is usually disturbed. The patient is apprehensive and anxious to a distressing degree, and his countenance exhibits a strong token of the existence of these feelings, especially in very malignant cases, when there is an expression of apathy, or one indicating a sense of horror or intense agony. In most cases, there is some confusion of intellect attended with constant pervigilium, though without so much derangement of the reasoning faculties as to amount to decided delirium. In some cases, however, the latter symptom assumes a more marked character, the disturbance of the mind reaching to the degree constituting mania, attended with wild or fiery looks and uncontrollable agitation of body. In other instances, there is a greater or less degree of stupor, through which, as Dr. Wood remarks, when short of coma, the signs of distress show themselves as through a veil. In not a few cases, though particularly in young persons of both

sexes, and in those of one of the sexes at two different periods of life, hemorrhages take place from one or both nostrils during the afternoon exacerbation.

This stage or period of febrile reaction continues with little or no mitigation during a time of longer or shorter duration, varying from some hours to two or three or more days; the duration being generally in inverse ratio to the violence of the attack. Having run this course, the fever subsides, never more, or very seldom, to return—the disease being one of a single paroxysm—and is followed by a state of remission or metaptosis. A great apparent amelioration of almost all the symptoms is now experienced. The skin, if hot before, assumes its normal temperature, or becomes much cooler than it was. The pulse returns nearly or quite to its natural state, falling to seventy or eighty pulsations, or even less, in a minute, and seldom if ever reacquiring frequency or activity and strength even during convalescence, when recovery takes place. The respiration at the same time becomes calm; gastric irritability, with the distress attending it, disappears more or less completely. The delirium or intellectual disturbance, if it existed; the headache, and the pain in the back and limbs, if not previously removed, subside. The patient, thus relieved from the bodily suffering and mental distress under which he had laboured, fancies himself well; is cheerful and hopeful; sits up in, or gets out of, bed. Sometimes, indeed, the result is favourable; the after-symptoms do not make their appearance, and convalescence is at once established. The eyes and face become tinged with yellow, or copious evacuations of bilious matter take place by stool, or a gentle or profuse perspiration sets in, or often, without any such critical signs, the patient speedily recovers—the disease consisting, in such instances, of but one of the stages into which it is usually divided.

Most usually, however, matters take a different turn. The amelioration described proves delusive, and its occurrence the harbinger of a dangerous condition. The great struggle between life and death is yet to come. The physician soon learns how little he can trust to the state of amendment he notices; for while in some cases a restoration to health soon follows, as we have seen, the cessation of the symptoms of the reactionary stages, in another and larger number of cases, he finds that this improvement is only temporary, and is soon succeeded by other phenomena of a more formidable character. Indeed, he discovers, on close inquiry, that during the very period of tranquillity which buoyed up the hopes of the patient, symptoms denoting the existence of undiminished danger existed. The tenderness of the epigastrium is unrelieved, and, in some instances, is even greater than it was before. The redness of the eye and the flush of the face have given way generally, but not always, to a yellow or orange colour, which gradually extends from the forehead and eyes to the face, neck, chest, and at last diffuses itself more or less completely over the whole surface. The urine also is found tinged with the same yellow hue. The pulse is sometimes slower than in health, and in bad cases the patient betrays a little heaviness of intellect or stupor.

This state of extraordinary, but imperfect remission, varies in point of duration from a few hours to twenty-four or longer. At the expiration of

that time, the symptoms already existing become aggravated, and to these, others are added. In some, but not many, the pulse becomes quick, irregular, and feeble. In a greater number of instances, it remains natural or slower than in health, and becomes still slower as the disease advances, until at last the pulsations are reduced to forty or thirty in a minute, and are withal feeble and irregular—the heart, at the same time, even in the most malignant cases, being found often to beat with considerable energy, and to continue doing so after the pulse has ceased to be felt at the wrists or other parts. The tongue becomes loaded—particularly in the centre—moist or dry, with or without redness of the edges. Thirst increases, and is often insatiable; nausea and vomiting, attended with heat in the stomach, appear again, and become constant—the matter ejected being mixed with streaks or flakes of a red or brownish colour. The respiration quickens, or becomes embarrassed; the skin becomes cool, dry, and parched; the anxiety at the præcordia is now distressing, accompanied with a sobbing kind of sighing, constant hiccough, and occasionally with an expression of deep anguish and despair.

In many cases, the mind remains clear and undisturbed; the irritability and extreme distress of the febrile stage is replaced by an extraordinary degree of apathy, and the countenance conveys the expression of resignation or indifference as to the issue. In other instances, the patient at this time is affected with a kind of cheerful delirium, imagining himself well. In others, again, though not delirious, he remains a long time as if in a deep reverie; and when aroused from this state, starts with surprise, and answers in a hurried manner. Many, on the other hand, while apparently in great distress, answer that they are well. As the disease progresses, this condition of mind is often succeeded by another of a different kind. Coma now supervenes, from which the patient is aroused by the act of vomiting, or by dreams. When thus aroused, he fancies himself perfectly restored to health, attempts to get out of bed, but soon relapses into a state of insensibility. In many instances, debility is more or less considerable. In not a few, however, the patient retains, to a late period of the disease, his muscular strength—to such an extent, indeed, as to get out of bed, and walk about his room, and further, if permitted. The physiognomy is peculiar and striking, and, as is well remarked by Dr. Jackson, conveys at once an impression of the malignant and dangerous nature of the disease.

It is not uncommon to find the tongue becoming, after a short time, cleaner and moister than before. Ordinarily, however, its foulness increases. It is sometimes tremulous, and with difficulty protruded, and when the patient succeeds in showing it, he not unfrequently forgets to put it in again. It is brown and dry in the centre, or smooth red, chapped, with sordes on the gums, lips, teeth, and nostrils; or, again, white on the edges, with a black streak in the middle. The vomiting, as the case advances, becomes less frequent, without, however, being accompanied with diminished irritability of the stomach, which rejects everything introduced into it. But while the frequency of the vomiting, when not excited by drink, somewhat diminishes, the quantity thrown up at each ejection increases—sometimes to an extra-

ordinary extent, the act of emission, which affords momentary respite to the patient, being effected by a sudden contraction of the stomach and other parts concerned, which propels the contents of the organ to a considerable distance. From this condition, recovery not unfrequently takes place—generally by a gradual receding of the symptoms, but sometimes by an evident critical revolution—the pulse acquiring force and activity, the skin becoming warm and moist, the irritability of the stomach lessening, and finally disappearing.

But in other cases, the disease progresses and presents symptoms of a still more formidable character. The matter vomited now consists of brown, blackish, or chocolate-coloured flakes or particles, diffused in a colourless liquid which, though at first slightly tinged by them, ultimately becomes black and opaque, resembling coffee-grounds floating in a serous fluid. In some cases, grumous dissolved blood is thrown up. The matter vomited is acrid—often excoriating the throat, tongue, and lips. Usual, however, as the occurrence of such vomiting may be at the present period of the disease, cases occur in which it does not take place, and the patient dies without having thrown up black matter. Be this, however, as it may, its appearance portends the most imminent danger, for though some recover after the discharge, the number is very limited. The abdomen is soft, seldom meteorized, but the stools, when they occur at this time, present much the same character as the matter ejected from the stomach, or assume the appearance of tar or molasses; or, again, consist of blood more or less pure. The urine becomes natural in appearance, or of a dark colour, and limpid. It is now often suppressed from deficiency of secretion, or simply retained. Sleep is interrupted, and attended generally with painful dreams. The face and breast become spotted as with ink. The jaundice—which, though so frequent an attendant of the disease as to have given it its name, is nevertheless often, especially in rapid cases, totally absent—becomes more diffused and of deeper hue. The skin is now often of a deep dusky yellow, or brown, mahogany bronze, or purple colour, imparting the idea of blood settled in a bruised part. The fluid becomes stagnant in the capillaries, forming petechiæ, vibices, or large blotches, and accumulates in depending parts and the extremities. It oozes, in many cases, from the nostrils, tongue, gums, anus, eyes, from leech-bites, blistered surfaces, and the punctures of the veins; and is, like all the blood in the vessels, dark-coloured and dissolved.

As the disease advances, the symptoms described increase in intensity, and are combined with others imparting a still higher degree of malignity and danger. Hiccough now sets in, and is soon constant and accompanied with the hippocratic countenance, difficulty of swallowing, and slow and stertorous convulsive respiration. The pulse becomes small, feeble, intermittent, and finally fades away. The alvine evacuations are highly offensive, of cadaverous smell, and, like the urine—which, if at all secreted, assumes a blackish and bloody appearance—are voided involuntarily. Subsultus tendinum not unfrequently follows; so also gangrenous spots; and, in a few cases, buboes, carbuncles and eschars in various parts of the body. Loss of speech, dimness

of vision, insensibility, low muttering, delirium, and coma, at times supervene; but it is not unusual to find patients retaining their intellectual faculties unimpaired to the last. Next, we have rattling in the throat, cold clammy sweats, cadaverous and peculiarly offensive odour of the body, cold respiration, the whole ending in death, which often occurs quietly, but in other instances in the midst of violent convulsions.

The duration of the disease varies, according to the nature of the cases, from three to nine days; sometimes it is shorter, at others longer; while, in cases of recovery, the convalescence is usually secure and rapid.

If now we pass from this general survey of the symptoms observed in the yellow fever, considered in the aggregate of the phenomena, to the several groups in which they usually present themselves, we shall find they are amenable to the two grand divisions or species, marked by two opposite conditions of the system mentioned—the one characterized by phenomena denoting an inflammatory vascular action, the other marked by those denoting a state of asthenia, impaired vascular action or congestion. Nor can we fail to discover, on closer scrutiny, that each of these species or general groups is susceptible of being subdivided into several varieties or grades, depending on different degrees of intensity, merging, undoubtedly, to a greater or less extent, into each other, and presenting phenomena common to all, but in many respects sufficiently distinct to enable us to arrive at a correct diagnosis, and to discover suitable therapeutic indications.

The first of these species—the inflammatory—will be examined under the heads of intense, mild, and ephemeral; the other—the congestive—under those of aggravated, adynamic, walking, and apoplectic.

INTENSE GRADE.—Cases of this grade of the *inflammatory* species, are usually, though not necessarily, ushered in by a chill, preceded by the premonitory symptoms enumerated above. In some patients, there is only a sense of coldness; in others, complete rigors—the attack commonly coming on at night, and the chill, when it occurs, often alternating with flushes of heat. This is succeeded by febrile reaction, which generally attains considerable intensity. The pulse becomes quick, frequent, strong, and full—beating from 90 to 120 strokes in a minute. The skin is hot, and, though sometimes moist, is usually dry and parched. The temporal and carotid arteries throb and beat with considerable force; the face is highly flushed; the eyes are remarkably red and bloodshot, the balls often appearing to be a mass of vessels gorged with blood. They are, besides, hot, painful—feeling as if they contained sand—and withal brilliant, shining, and watery, while the lids are sometimes swollen. The patient complains of intense pain in the supra-orbital region, and severe rachialgia, and suffers, besides, in the thighs and legs. The tongue is moist, covered with a white or yellow fur, and usually of a crimson red at the edges and apex. The gastric organs soon share in the morbid derangement. There is a sense of anxiety, constriction, and intense heat at the præcordia. Nausea soon supervenes, increases rapidly in intensity, and terminates in retching and vomiting. The stomach is often

distended, and, in some cases, painful on pressure, and generally affected with an almost unconquerable irritability. These gastric symptoms, though sometimes early developed, are not usually so, to any extent, until from twelve to twenty-four hours after the outbreak of the attack, when they assume a permanent character, and constitute a source of great distress and suffering. The matter thrown up consists of drinks, and other articles swallowed, mixed with a clear, glairy mucus, and occasionally, with a substance of a sea-green colour and bitter taste. In a few instances, bile is ejected, and appears to consist in that which was contained in the gall-bladder or duodenum at the moment of the attack. The urine is deficient in quantity, high-coloured, and often sedimentous. The bowels are ordinarily costive, and hard to move; the matter evacuated being at first soft and feculent, and sometimes tinged with bile; then, when no cathartics are used, becoming of lighter colour, and of starchy, cream-like appearance. The patient suffers from restlessness and jactitation; moans, sighs, shifts his position constantly, and feels a disposition to leave his bed and walk about. The respiration is laborious and hurried, and the countenance presents an anxious, gloomy, sad, or impatient expression. The patient is apprehensive and anxious to a distressing degree. His intellect is confused, and he experiences constant pervigilium. Delirium, properly speaking, seldom occurs. In some cases, however, it makes its appearance, reaching, at times—even from an early period—to the degree constituting mania.

This stage of inflammatory reaction continues, with little or no mitigation, from some hours to two or three or more days—generally from sixty to seventy-two hours—and is succeeded by the state of remission, the metapto-sis or stadium without fever, already described. The pulse loses its febrile excitement, and becomes almost natural, or even slower than in health; the skin regains its natural temperature; the pain of the head, loins, and extremities disappears or is greatly diminished. The confusion or derangement of mind likewise subsides, and the patient either thinks himself, or endeavours to represent himself, well; is cheerful, sits up in, or gets out of, bed, and expresses an appetite for food. The redness and glistening appearance of the eyes are no longer apparent, but are replaced by a yellow tinge of the adnata. This state of relief, which proves but too frequently delusive—few cases ending at this time—continues from a few hours to twenty-four or thirty, and gradually glides into the succeeding stage. Prostration follows; the pulse becomes rapid, irregular, and depressed—or, as is more usual, it is of natural frequency, or even slower than in health. The tongue becomes loaded with a brown fur, presenting a darker streak in the middle, and is swollen and moist. As frequently it is clean, with a slight, pasty coating, or of a deep fiery red; occasionally, in an advanced period, it is bloody or dry, black, and chapped, with dark-coloured sordes about the mouth, lips, gums, and nostrils. Respiration quickens and becomes laborious; thirst increases, and is often insatiable. The anxiety at the præcordia becomes distressing, and is accompanied often with hiccough and sighing. The pain at the epigastrium increases, and, in many instances, becomes intolerable. It is increased during

vomiting, which is now effected without difficulty, the contents of the stomach being spouted out, sometimes to a considerable distance, by a sudden contraction of the stomach and abdominal muscles. The matter vomited now consists of brown, blackish, or chocolate flakes or particles diffused in a colourless liquid, and gradually acquires in fatal cases the qualities characteristic of the black vomit already described. In some cases, the bowels give passage involuntarily to black, acrid, and offensive discharges, sometimes having the external characteristics of tar or molasses; at other times consisting of blood. The jaundice, which had commenced towards the beginning of this stage about the forehead and eyes, next extends to the face and chest, and at last covers the whole surface. It gradually acquires a deeper hue, the skin being of a deep husky, or of the brown, mahogany, bronze colour mentioned. In many instances, however, it is absent or restricted to the adnata, or shows itself only after death. In many cases, the mind is clear and undisturbed, and remains so to the last; the patient not unfrequently exhibiting an extraordinary degree of apathy, with an expression of resignation and indifference. In other individuals, we notice the various modifications of delirium enumerated. In many instances, the debility is more or less considerable; but in as large, perhaps a larger number, the patient regains his muscular strength, if he had lost it before, and retains it often to the last to an almost incredible degree. As the case advances, the body becomes cold and clammy; the urine is blackish or bloody, and often voided involuntarily. More frequently it is suppressed or simply retained. Hemorrhages take place from all the natural outlets, the blood being dark and dissolved, and death, preceded by intolerance of light, petechiæ, meteorism, singultus, eructation of offensive gas, subsultus tendinum, convulsions or coma, closes the scene. The rapidity of the disease, in this grade, is generally proportioned to the violence of the inflammatory action of the second stage; the latter, in its higher degree, overwhelming and disorganizing rapidly the vital organs, and thereby hastening the occurrence in them of changes incompatible with the continuance of their functions, and, as a necessary consequence, with the continuance of life.

That death is not the inevitable result of this grade of the inflammatory species, I will not deny. But recoveries do not often occur in the most violent cases, and when they do, they occur before the accession of the black vomit and succeeding symptoms, and are effected by a gradual amendment of the symptoms, sometimes through the instrumentality of some critical movement.

MILD GRADE.—In this grade of the inflammatory species, the symptoms are much the same at the outset as those just described, though less violent, rapid, and tumultuous. The disease presents similar premonitory symptoms, and is as often preceded by chills or rigors. The pulse is strong, full, and frequent, between from 90 to 120 or more in a minute, or it is comparatively corded and hard; the skin is hot and dry, sometimes soft, with a disposition to diaphoresis. The pain in the orbits, back, and forehead is intense; the face is flushed, attended at first with cerebral excitement, producing occasionally

a flow of spirits, amounting almost to slight intoxication; stupor or delirium sometimes supervenes; but in many cases the intellectual functions are unimpaired. Respiration is hurried and often laborious; the tongue is slightly furred and moist, with more or less redness at the edges and apex. As the disease advances, this organ often becomes dry and covered with sordes, and assumes sometimes some of the other appearances described under the preceding head. The uneasiness and sense of heat at the præcordia are very troublesome; the thirst and desire for cold drinks great; nausea and vomiting are distressing, and with difficulty controlled; the bowels give passage to dark-coloured and offensive discharges. This period of excitement continues, as in the former grade, from a few hours to two or three days; but, unlike what takes place in that grade, it is attended with more decided exacerbations and abatement of the fever—making, in some instances, an approach to decided remissions—and often extends to four or five days. It is then followed by the aforementioned state of remission, which is also more perfect, and in many cases the harbinger of recovery—convalescence dating from the subsidence of fever which takes place at this period, and the disease, which, in such cases, consists of only the first stage, going off with bilious critical discharges by the bowels, a moisture over the skin, or diaphoresis, a copious emission of urine, or a hemorrhage from the nose—with or without jaundice—often without any evident crisis. In other cases, however, this state of metaptosis proves delusive, and is succeeded by many of the symptoms enumerated as constituting the closing stages of the preceding grade. In some cases they assume a character of great malignancy, and if not arrested by art, or the recuperative efforts of nature, carry off the patient in the manner mentioned. In others, they stop short of the black vomit, and the patient is gradually restored to health by the intervention of art, seldom through the unaided efforts of nature. In others, again, though in very small number, the patient is saved even after the supervention of that and other usually fatal symptoms. But, whatever be the apparently visible character and issue of these cases, they evince the fact that the yellow fever, in this grade, is evidently less malignant in its nature, and within the range of remedial agencies. The bowels are acted upon without much difficulty by cathartics or enemata, and the operation is productive of relief; the pain and affection of the head and other parts are under the control of proper depleting remedies, general and local, of revulsives, &c.; the gastric irritability, though obstinate, is not always as difficult to manage as in the preceding grade, and diaphoresis is generally easily obtained by external and internal means.

EPHEMERAL GRADE.—The disease in this grade is mild and manageable. It is, as may be presumed, of short continuance, terminating sometimes in a single day from the action of proper and even mild treatment. In other cases, it continues from three to five days; in which event it is sometimes attended with slight and imperfect remissions. But, although mild, and easily under the control of art, and often removed by the unaided efforts of nature, the disease in this grade presents the same features, the same outline

of phenomena, as characterize the other two grades, and enable us to establish a correct diagnosis.

Like the others, it is usually preceded by a chill, while the period of reaction is attended with the pain in the supra-orbital region, in the loins, the joints and limbs, noted in the other grades; but, in the one under consideration, these symptoms are not so intense. The eyes present the suffused, shining, and glistening appearance described; the face is flushed; the skin hot, dry, and harsh to the feel; delirium seldom attends, but the mind is usually confused; the urine is scanty and high coloured; the respiration is sometimes embarrassed; the pulse is quick, frequent, and full; the tongue is often loaded and red; and the thirst is sometimes considerable. In the milder cases, the stomach generally remains undisturbed, or is but slightly affected. In the more severe cases, however, nausea, and irritability of the stomach, and copious vomiting are constant attendants; the matter ejected consisting of the ingesta mixed with mucus, and of bile—the quantity of the latter being much larger than in other grades of the disease. Those symptoms which exhibit evidence of an open and well developed febrile paroxysm, subside sometimes suddenly, at other times gradually, the crisis being marked by increased alvine evacuations, by diaphoresis, or epistaxis.

CONGESTIVE SPECIES. 1. *Aggravated Grade*.—Dangerous, and frequently fatal as may be the first grade of the inflammatory species of the yellow fever, more apprehension must be felt when the disease assumes the congestive form. In this, whatever be the grade it presents, the efforts of art prove but too frequently of little avail, those of nature are powerless, and the large proportion of individuals thus attacked are doomed to almost certain death. In the inflammatory form, we have seen that the disease—especially in its milder grades—consists sometimes of but one of the ordinary stages, convalescence commencing at the period of remission; and that in other cases it consists of the first, second, and portions only of the third stage, the patient recovering before the accession of adynamic symptoms. In the congestive species the disease passes, as it were, immediately from the first sign of indisposition to the last stage, without going through that of reaction. In this grade, the attack comes on suddenly, and is attended from the outset with considerable prostration—quickly followed, in many cases, by giddiness, stupor, almost unconquerable disposition to sleep, loss of memory, and a desire to be left alone. Together with this stupor there is a sense of weight and oppression, rather than acute pain, in the head. In a few cases there is delirium, either transient or ending in confirmed coma. The face is pale and purplish, or livid in colour, with an expression of countenance either stolid or apathetic—the patient appearing in a taciturn mood, and uttering no complaint. In other instances, he is entirely insensible, with his eyes wide open, and presents, at times, an expression indicative of a feeling of distress, horror, and even intense agony. The acute pain in the loins and extremities, noticed in the inflammatory species, is replaced by one of an obscure character, and a feeling of helpless debility about the spine—most distressing

about the sacrum—sometimes attended with a paralytic failure of the lower extremities. The eyes have a dull, red, glassy, or drunken, idiotic look—with, in some cases, a dilatation of the pupils, and sleepy motion. The state of the skin varies in different cases, but is always deficient in tone—dry, dense, or unctuous, or sometimes covered with, and as if melting in, sweat. It is generally cool—sometimes cold, except at the centre portion of the body, which is hot; in some instances smooth and white, and occasionally loses more or less completely its sensibility and irritability. The pulse varies considerably in regard to both frequency and expansion, being sometimes accelerated, at others not more frequent than in health; sometimes full, at others small. But it is always weak, and offers no resistance to the pressure of the finger; and is occasionally almost wanting at the wrist, though at the time the heart and carotids may be throbbing forcibly. As the disease advances, it diminishes in frequency, the beats not amounting, at times, to more than forty, or even thirty in a minute. In some cases it becomes intermittent. When blood is drawn, it is generally found dark or discoloured, and seldom retains its natural character.

During this time, there are tenderness of the epigastrium, and tension of the hypochondria; weight and oppression at the præcordia. The stomach becomes early irritable; vomiting soon follows—the matter ejected rapidly assuming the character of the deadly black vomit. The discharges from the bowels are scanty, cream or clay-coloured, puruloid or gelatinous; sometimes they are of a pea-green colour, or black and bloody; the respiration is laborious. The tongue is sometimes natural, at others it is at first pasty, with patches of white fur, its edges and apex being red. Occasionally it appears as though scalded with a hot iron. It is often tremulous, and when the patient puts it out he often forgets to draw it in again. In some cases, the tongue becomes dry, the papillæ being at the same time separated by deep fissures. Besides these, we have orthopnoea, sore throat, deep and interrupted sighs, hemorrhages of dissolved blood from some one or more of the natural outlets, a yellow or bronze colour of the skin, suppression of urine, extreme restlessness, low monotonous wailing, and other symptoms denoting the utmost danger or the approach of death.

In some cases of this variety, the disease is principally characterized by an overwhelming oppression at the præcordia, attended with slow, laboured respiration, deep sighs and groans. In others, the stomach is the organ most implicated, the patient being affected with constant vomiting and intense epigastric distress; the whole soon followed by black vomiting and death. In a different set, again, the pulse is nearly natural, the tongue clean, and the stomach calm. But this is accompanied with excessive restlessness, and great anxiety and distress, soon followed by black vomit and fatal collapse.

The symptoms above enumerated are characteristic of the more intense form of congestive yellow fever, from which but a very limited number of those attacked recover. It may not be improper to remark that, in some instances, the disease, though marked by the same train of phenomena, assumes a less formidable character, stops short of the black vomit and other fatal symptoms, and proves comparatively mild and manageable.

2. *Adynamic or Typhoid Grade*.—This grade, which occurs in persons of deficient vital power, or under circumstances tending to foster or develop the typhoid diathesis, is usually ushered in by a sense of chilliness, which is succeeded by one of burning heat, partially distributed over the body, and affecting principally the under parts of the arm, and inner surface of the thighs. The circulation is depressed; the pulse being small and weak. The eyes present a dingy appearance. The head is severely painful, and attended with confusion of thought and dimness of vision. The skin assumes an olive hue, and is covered with petechiæ or vibices. Hemorrhage from the natural outlets, leech-bites, &c., follow; as also excoriations about the nose, mouth, or other parts; gangrene of blistered surfaces; sometimes anthrax, buboes, and more frequently venous infiltration under the skin, or in the interstices of the muscles.

3. *Walking Grade*.—In this grade, the functions of organic life appear to be at first alone implicated—those of animal life remaining almost untouched. The patient, though sometimes in bed, is found more frequently sauntering about his room; and, indeed, he at times walks about the street, for recreation or business; and though, in some instances, he states that he is weak, in others he exhibits at intervals, or throughout, marks of considerable muscular strength. He complains of nothing, denies his being ill, amuses himself in reading or otherwise, and, to a casual observer, appears to be slightly, if at all, indisposed. To the physician, however, matters appear in a different light; for he may generally observe that the patient exhibits an unusual expression of countenance—dull and listless. The eye is watery; the complexion is almost of a mahogany colour; while the pulse is found to be exceedingly weak, and even totally absent. Black vomit overtakes him even while occupied in the way mentioned, or very soon after, and death speedily ensues.

4. *Apoplectic Grade*.—In some of the cases classed under this head, the patient is struck down suddenly, as if by lightning, with stupor or coma, and death, preceded by convulsions, soon follows. In other instances, the progress is less sudden. Without even the slightest premonitory symptoms, the patient is in an instant seized with vertigo and confusion of mind. He complains of dull pain and fulness of the head, together with spasmodic pain and considerable debility in the legs; coldness, debility, and a feeling of uneasiness in the spinal region. The pulse varies in different cases in point of fullness and frequency, but is always weak, and finally becomes faltering. The skin is cold, sometimes dry and flabby, but generally unctuous or bedewed with cold perspiration; the stomach is sometimes irritable. In the mean time, the patient lies as if stunned, with dilated pupils and an expression of gloom on his countenance. From this unpromising state an effort at reaction occasionally takes place; but this scarcely ever leads to a successful result. More generally, the patient becomes perfectly comatose, the eyes assume a glassy appearance, the pulse fades away, involuntary discharges and profuse hemorrhage supervene, and death soon ensues.

CHAPTER IV.

THE YELLOW FEVER THE SAME EVERYWHERE, IN TEMPERATE AND TROPICAL CLIMATES—SEPARATE EXAMINATION OF ITS PHENOMENA—PREMONITIONS—MODE OF INVASION.

IN the last chapter, a full, perhaps unnecessarily minute, description was given of the symptoms of the yellow fever as it has appeared in Philadelphia at various periods from the early settlement of the city to the year 1854. If, with these details fresh in his memory, the reader revert to the history of the disease which, under the same name, has long desolated, and continues to desolate many sections of our country; which has on several occasions visited the cities of Spain and Italy, and is viewed as endemical in most parts of tropical regions; if he peruses the descriptions of the fever for which the professional world is indebted to Moultrie, Hosack, Dickson, Arejula, Pariset, Gillkrest, Palloni, Bally, Warren, Williams, Desportes, Hillary, Bancroft, Jackson, Rochoux, Wilson, Moseley, and many others that might be named, he will acquire the most convincing evidence of the perfect identity of that fever with our own.

That a somewhat different opinion has been occasionally ventured upon at various periods—from the days of Arejula to the present—is a fact well known to most readers. As regards the fever of this country generally—from Boston to Galveston—its identity with that of Philadelphia has never, so far as I have been able to discover, been contested. In all places, we discover that the disease is one of a single paroxysm. In all, we notice the characteristic appearance of the eye and countenance; the lull or stadium without fever of the second or third day. In all, we find the peculiar absence of febrile excitement after the subsidence of that stadium; the same gradual slowness of the pulse and depression of cutaneous heat in the after stage; the same coloration of the surface; the same character of the matter ejected from the stomach. While such is the uniformity of sentiment in relation to the analogy of our yellow fever with that of other parts of this country—a uniformity fully borne out by the writings of Brown, of Boston; Hosack and Miller, of New York; Davidge, Potter, and Reese, of Baltimore; Moultrie, Lining, Irvine, and Dickson, of Charleston; Barton, Thomas, Gros, and Girardin, of New Orleans; Smith, of Galveston, and others too numerous to be mentioned in this place; while, I say, this sameness of sentiment prevails on the subject in question; and while, moreover, little or no difference of opinion exists as to the identity of the yellow fever of North America with that which at times has prevailed in Europe, as we easily gather from the writings of Arejula, Fellowes, Pym, Jackson, Doughty, O'Halloran, Rochoux, Pariset, &c., which clearly show that the latter is marked by precisely the

same pathognomonic phenomena as the former, there are not wanting some who, differing from the majority of our best authorities, affirm that this fever differs essentially from that which in tropical regions is designated by the same name. On the subject of this dissimilarity much has been said, and considerable ingenuity and talent displayed; by none more strikingly than by Dr. Rochoux, the most zealous, best informed, and most pertinacious defender of its reality. To those, however, who investigate the subject attentively, it will without difficulty appear that the success obtained is far from being commensurate with the efforts made to establish the existence of a fundamental difference. It is true, that, if, limiting our examination of the character, phenomena, and mode of progression of the West Indian fever, to this disease as described in the large and interesting volume which that distinguished author has put forth in defence of his peculiar views, and in another publication, devoted exclusively to that fever, we compare it with the description he has furnished of the fever of Barcelona—a fever he regards as identical with that of Philadelphia and other parts of extra-tropical latitudes, and which, in order the better to distinguish it from the former, he denominates amaril, or yellow typhus—we might, perhaps, feel disposed to assent to his opinion; for, in the portrait therein drawn of the West Indian fever, we discover a disease of an open inflammatory character, marked by phenomena indicative of the undoubted existence of gastro-enteritis, and the usual symptoms exhibited in such cases everywhere; while, in the description he gives of the amaril typhus, we note a complaint of a diametrically opposite character, showing a strong tendency to a malignant or adynamic depression, and many other phenomena either not mentioned as appertaining to the yellow fever of tropical regions, or contrasting in a greater or less degree with the corresponding ones noticed in the latter. But if, with a full knowledge of what has been written on the fever of temperate regions, not by Mr. Rochoux alone, but by our own writers and others in Europe, we open the principal works on tropical yellow fever, we shall find that matters present a very different aspect. Consult the volumes of Pouppé-Desportes, Warren, Chisholm, Jackson, Clark, Dariste, Savarési, Imray, and many others, and it will be plain to the commonest understanding that, in assuming the form of fever which he had occasion to see at Guadaloupe during a single epidemic, as the true and constant type of the disease, Dr. Rochoux has been guilty of a great oversight; inasmuch as, during some seasons, and under peculiar circumstances, the fever of those regions presents an aggregate of phenomena, a diathesis, and a mode of progression, identical with, or closely allied to, those observed in the fever which he represents as belonging exclusively to regions beyond the tropics. So true is this, that the description of several West Indian epidemics on record—the memorable fever of Grenada in 1793, as described by Chisholm and Steward; that of Dominica, in the same year, by Clark; the description of several of the varieties noted by Jackson, Ralph, Imray, Savarési, Lemprière, not to mention others of as easy access, might, with very trifling changes, serve more appropriately for the epidemic of Barcelona in 1821, of Cadiz in 1800, and some of those of

our own cities, than for the disease which is stated by Dr. Roehoux to prevail exclusively in the Antilles. Dr. Jackson, who, after investigating the fever in the West Indies during some thirty years, and describing its various modifications minutely and with consummate ability, witnessed the epidemics of Cadiz and Xeres in 1820, found, as has already been stated, that the disease in those cities presented the characters he had noticed on various occasions in America, and entertained in consequence no doubt of their identity; and from all we have seen of our own fever, it may safely be affirmed that the same conclusion will be drawn by those who, while familiar from personal observations with the various modifications it presents at different times, will take the trouble to investigate the disease as it shows itself within the tropics.

But, even could we not appeal to the testimony of the high authority referred to—a testimony which might be strengthened by that of Pym, Gilpin, Fellowes, Chervin, O'Halloran, Bally, Doughty, and others, who witnessed the fever in both hemispheres, and admit its identity everywhere; even were it justifiable to base an opinion respecting a question of this kind on the result of observations made during a single epidemic in the different latitudes mentioned; even were the differences as great as represented by Dr. Roehoux; and even were we unable to point out instances in which the description of certain epidemics in the West Indies might with little change be applied to the Barcelona disease, which Dr. R. regards as the type of his amaril typhus, the correctness of his opinion might still be contested; for it is easy to discover, amid cases similar to those he regards as alone exhibiting the features of the true yellow fever of tropical climates, instances—sometimes in large number, sometimes otherwise—in which the disease presents the form or character which, according to him, appertains to the other. In other words, in the course of an epidemic in the West Indies, where the fever exhibits a train of phenomena different from that supposed to be observed in temperate regions, and along with cases of that description which, in ordinary seasons, when the disease presents itself sporadically, attacks only the unacclimated, instances occur in which the fever bears a strong analogy to that which, in the opinion of Dr. Roehoux, is noticed beyond the tropics. In fact, the same differences exist among individual cases of fever prevailing in the tropics—sometimes the one form, at other times the other having the ascendancy—as are exhibited between those described as examples of the disease in the two regions. It follows, as a necessary consequence, that if a disease similar to the one existing in temperate climates exists also in the West Indies and other kindred regions; that if the former differs from that represented as the true type of yellow fever, and that if the two diseases prevail in the same place not only at different times but also simultaneously, there can be no ground for the distinction attempted to be drawn between them.

Nor is this all. While such differences are noted in warm latitudes, the same results obtain in this country, where, although epidemics in which the disease bore a strong resemblance to that of Barcelona have occurred, others, in which it was no less analogous to the disease assumed as characteristic of

true yellow fever, have been described by our highest authorities. Nay, on some occasions when the first mentioned form had the ascendancy in point of numbers, instances of the other form have been encountered. Of the truth of this statement respecting the varieties of form assumed by the fever of temperate regions, and of the approximation of this disease to that described by Dr. Rochoux as occurring in the West Indies, the details entered into in the preceding chapter, relative to the classification of the cases and the description presented of the fever of this city, afford ample proof; while a survey of the result of observations made in other parts of the United States will enable us to arrive at a like conclusion. In Europe itself, judging from the descriptions of Jackson, Palloni, Arejula, Berthe, and O'Halloran, the inflammatory form, even in its higher grades, as well as the force and persistence of reaction, presented itself, if not as frequently as it has at times done on this side of the Atlantic, at least sufficiently so to show that it is not to be encountered exclusively in the West Indies, and that its absence cannot be adduced by Dr. Rochoux in support of his opinion.

From the whole of what precedes, and an examination of the varied information we possess in reference to the yellow fever generally, we must be prepared to admit either that there are two yellow fevers in both temperate and tropical climates, or that the disease presents in such climates as great differences in its character and the general train of its phenomena at different seasons, in the same place, and often in different places during the same season, as those adduced in support of the distinction affirmed to exist between the fever of tropical and that of temperate zones; for, contrary to the representation of Dr. Rochoux, there is scarcely a single phenomenon manifested in the one which is not observed in the other at some time or other. Indeed, in the description of the very disease pointed out as differing from that of the temperate regions, we find an enumeration of those phenomena and peculiarities which, whatever be the modifications the disease may present, are viewed by every one and everywhere, as pathognomonic of it—the red, vultuous, shining aspect of the face; the peculiar and intense injection of the conjunctiva; the brilliant, watery appearance of the eyes; the anxiety; restlessness; watchfulness; acute supra-orbital pain, and all the symptoms indicative of a febrile paroxysm, succeeded by the metaptosis described; the absence of fever in the after-stage of the disease; the jaundice, black vomit, passive hemorrhages; the suppression of urine, and the frequent integrity of the intellectual faculties; the retention of muscular strength, and other symptoms which I have noted in the preceding chapter.

It need scarcely be remarked that, in investigations of this kind, pathognomonic symptoms—such as establish the individuality, as it were, and the nosological position of the disease—should alone be assumed as objects of comparison; because, apart from these, it is found that in the group of phenomena observed in various cases there are many, the presence or absence of which does not affect the question of the character of the disease, and which owe their origin to a variety of circumstances not necessarily connected with its cause. This fact, which is true as regards epidemic diseases generally, is

particularly so in respect to the yellow fever, and, if not attended to in an inquiry like the present, might lead to a very erroneous conclusion. Though upon this point I have already entered at large, I must be permitted to repeat that the accounts extant of the various visitations of that fever in the same country, our own, for example, show that in some seasons various symptoms manifest themselves frequently which, at other times, are comparatively of rare occurrence. The diathesis varies at different periods; particular organs are more or less affected, &c.; but the disease does not on this account cease to be precisely the same at these different times; for, amid the diversified phenomena noticed, there are some always present which impart to the disease a stamp of individuality, and enable the physician to distinguish it from all others. The same difference which at various seasons occurs among groups of cases, occurs in the same seasons among individual cases. It often happens that patients, lying in the same wards of an hospital, labour under symptoms so variously grouped as to lead an inexperienced practitioner to conclude he has before him several diseases bearing little affinity to each other; while to one more conversant with the subject, the family connection of all the cases is rendered apparent by the immediate detection, in each, of those pathognomonic signs which serve to identify the disease.

It must follow that, in comparing the diseases of different countries, the same exclusive regard must be paid to the essential and fundamental phenomena, as in comparing the complaints of different seasons in the same country; for, if the secondary or accessory symptoms vary at different times in the latter, they may be supposed, with greater reason, to vary in distinct latitudes, although the disease remains identically the same. An examination of Mr. Rochoux's chapters on the distinction between the yellow fever and the amaril typhus, founded on the descriptive history of the two diseases, will, I think, show that he has not had sufficient regard to those considerations; laying stress, to attain his end, on various symptoms and phenomena which are merely accessory, and not essential to characterize the disease, and which vary as often in the several epidemics of the same as in that of different countries, and at the same time representing some as serving to distinguish his true yellow fever from the amaril typhus, which, so far from belonging exclusively to the former, have been noticed more or less frequently in the latter.

Assuming, from a consideration of these facts, the identity of the yellow fever, in whatever place it may present itself, I next proceed to a separate examination of the phenomena of the disease as it occurred here, as also of various points connected with its mode of progression and its results, as well as of the source and nature of the most characteristic of those phenomena. In the progress of this examination, the identity in question will become more apparent.

A. *Premonitions*.—Having, in the preceding pages, presented a full *exposé* of the symptoms of the yellow fever, such as it usually exhibits in its progression through various stages from the outset of the attack to the close by death or recovery, and offered a similar tableaux of the several groups

of phenomena constituting the different forms or varieties into which the disease may be divided, I pass to the consideration of each individual symptom, and to a comparison of those noticed here with the corresponding ones observed in the yellow fever of different sections of this country and of other climes. In conducting this examination, it will be necessary to pass in review the morbid manifestations exhibited by each of the organs and tissues implicated in the disease, and the changes occasioned in the functions and products of those parts.

The phenomena, thus brought out in relief, may be divided into those which present themselves prior to the development of the changes constituting the attack, properly speaking, and which, for that reason, are denominated precursory or premonitory; and, secondly, those which, by their aggregation, impart an identity to the disease, indicate the nature and extent of the sufferings of the organs and tissues affected, and without which, indeed, the complaint could not have an independent existence. I shall commence with the former, and, after speaking of the mode of invasion, proceed to the consideration of the latter.

In the foregoing description, the reader will have perceived that the disease differed somewhat, in different cases, in regard to its premonitions; in some instances, being announced by hours or days of indisposition; in others, coming on suddenly, and with little, if any, warning. That such has been the case in the epidemics of this city, may be learned from Dr. Rush and our other writers, who point out the nature of the premonitory signs by which the fever has often been foreshadowed. "The precursors, or premonitory signs of this fever," says the former, in his account of the epidemic of 1793 (iii. 51-2), "were costiveness, a dull pain in the side, defect of appetite, flatulence, perverted taste, heat in the stomach, giddiness or pain in the head, a dull, watery, brilliant, yellow, or red eye; dim and imperfect vision; a hoarseness, or slight sore throat; low spirits, or unusual vivacity; a moisture on the hands; a disposition to sweat at nights, or after moderate exercise, or a sudden suppression of night-sweats." More or less of these symptoms frequently continued for two or three days before the patients were confined to their beds, and in some people they continued during the whole time of its prevalence in the city, without producing the disease (p. 52). In 1794, Dr. Rush observed two other signs—a frequent discharge of pale urine for a day or two before the commencement of the fever; and sleep unusually sound the night before the attack (iii. 203). In 1797, the attack, in several cases, was preceded by acute toothache; and, in one patient, there was an almost total inability to close the mouth, from soreness of the teeth. In some of these cases, mercury had been used to obviate the disease (iv. 70).

Dr. Currie (p. 19), in like manner, states that it was generally preceded by a slight indisposition—such as muscular debility, and a sense of lassitude on exercise or motion; drowsiness, slight pain and giddiness in the head, pain in the back and loins, defective appetite, flatulent eructations, &c.; and a like statement is made, though not in such detail, by Dr. Barnwell (p. 360),

Dr. Caldwell (p. 80), Dr. Deveze (p. 21), Dr. Wood (i. 83), and S. Jackson (pp. 50, 9).

But, as we have seen, the disease very often, if not generally, attacks abruptly in the midst of ordinary health, or is not preceded by perceptible indisposition. On this head, we may again appeal to the high authority of Dr. Rush, as well as to the writers cited, and to Dr. Nassy and others, who make no mention of the occurrence of premonitory symptoms. "Frequent as the precursors of the fever were," says Dr. Rush, "they were not universal. Many went to bed in good health, and awoke in the night with a chilly fit. Many rose in the morning, after regular and natural sleep, and were seized at their work, or after a walk, with a sudden and unexpected attack of the fever (iii. 52).

Very similar have been the results of observations made in the other cities of the middle and northern sections of this country—Baltimore, Norfolk, Wilmington (N. C.), New York, New Haven, Middletown, Charleston, and Savannah, where the disease is very often preceded by some one or another of the symptoms enumerated—general languor, lassitude, sluggishness, weariness, restlessness and heaviness, depression of spirits—sometimes approaching to stupor—or, unusual flow of spirits; disinclination for bodily exercise, debility, pain in the head, loins, and limbs (*coup de barre*), loss of appetite, nausea, unpleasant taste in the mouth, perspiration with creeping sensation, sighing, yawning, &c.; while frequently the attack comes on abruptly, and without premonition.¹

Dr. Heustis, in his Topographical View of Louisiana (p. 109), gives the following minute enumeration of the phenomena by which the fever of New Orleans is usually preceded: "Listlessness, want of energy, and disinclination to motion; and exertion of every kind becomes laborious and irksome. This sensation of languor and lassitude increases to a degree of anxiety and uneasiness; the mind is easily fatigued by the slightest attention, and exercise is difficult and painful. An uneasy sense of stiffness is perceived in the motion of the eyes, a degree of tension is felt across the orbits, and a dull, heavy pain in the head. The joints feel stiff; an uneasy sensation of weariness takes place in the small of the back; a soreness affects the muscles of the legs; the animal functions are debilitated; and indescribable anxiety, at times, pervades the system. For a short time he seems to stand tottering upon the isthmus between health and disease; flattering himself that his indisposition is slight, and that in the course of a day or two health will return without the interference of medical aid. By turns he thinks his disorder imaginary, and endeavours to partake of the passing amusements; he however soon finds himself fatigued, and so much indisposed as to be under the necessity of going to bed." Dr. Shecut, of Charleston, calls attention to the fact of a "peculiar and almost indescribable appearance of the whole physiognomy, of

¹ Davidge, p. 102; Drysdale, i. 36; Addoms, p. 10; Alex. Hosack, p. 11; E. H. Smith, p. 115; Munson, p. 179; Davis, p. 7; Barton, p. 15; Dalmas, p. 6; Harrison, p. 131; Townsend, p. 143; Archer, v. 66; Hill, v. 89; Tully, p. 294; N. O. 1820, p. 7.

the patient's expression of peculiar anxiety and alternate dejection, which is seldom found in autumnal remittents" (pp. 118-19).

Dr. Stone, of Natchez, noted a whirling sensation in the head (p. 556). Dr. Waring, of Savannah, besides some of the symptoms enumerated, notices a burning sensation in the stomach (p. 45). Dr. Cartwright (ix. 13), and Merrill (ix. 244), of Natchez, remarked in some cases an unusual exhilaration of spirits. The attention of Dr. Lining, of Charleston (p. 411), was directed to acute pain in the knees. Dr. Barrington (xii. 311) speaks of severe pain in the penis. Gros (p. 9), Thomas (81), the reporters on the fever of New Orleans, in 1820 (p. 71), and Girardin (p. 32), limit themselves to an enumeration of some of the phenomena mentioned; while most of the writers named, as well as Barton (p. 10) and Harrison (p. 131), represent the attack as commencing suddenly in many, if not all cases. To this I may add that Dr. Wragg, who had charge of the Roper's Hospital, in Charleston, during the epidemic of 1854, states that the manner in which the disease invaded was strictly inquired into in all practicable cases: "It was noted in 225, that, out of this number, 92 attacks were sudden; 32 came on insidiously, the patients complaining of malaise, &c., for a considerable time, and then gradually becoming feverish; and that 101 offered the usual symptoms characterizing the approach of fever."¹

The yellow fever of our country, therefore, whether of the Middle, Southern, or Eastern States, differs in nothing from that of this city, so far as regards the signs of indisposition by which it is preceded. Most of the phenomena noticed elsewhere have fallen under our cognizance here, and the few that may not have done so, were mixed up with others familiar to our practitioners, and constitute, at most, a trifling exception to a general rule. Even the minute enumeration, borrowed from Dr. Heustis, might apply as well to our fever as to that of Louisiana. Finally, everywhere, the fever very commonly comes on abruptly, and without warning.

In the fever of Spain and of Leghorn, we find a similar train of premonitory signs in many cases, and a like suddenness of attack in others. Berthe (p. 76), and Caisergues (p. 165), who have offered good and minute summaries of the symptoms of the epidemic of Cadiz in 1800, have done little else than repeat what had been said on the subject in question by the writers on the fevers of this country; and from the former we learn that the disease often attacked abruptly—*par explosion*. And the descriptions, for which we are indebted to Arejula (p. 169), Burnett (p. 5), Pym (p. 228), Pariset (pp. 369-372), Audouard (p. 55), Blin (p. 6), Velasquez (p. 9), Pariset (*Obs.*, p. 29), Gillkrest (ii. 270), Ratier (p. 55), O'Halloran (pp. 76, 122), Palloni (p. 2), Brignole (p. 19), Rochoux (p. 455), and Jourdain (v. 257), corroborate the statement made by other writers as to the character of the premonitory symptoms, and more particularly to the suddenness of the onset of the disease in a large number, on some occasions in the great majority, of cases.

Nor shall we find it different if, turning from temperate climates, we

¹ Charleston Journal, x. 71.

examine into what takes place in the yellow fever of tropical regions. "The yellow fever," says an intelligent writer who had in view the latter disease, "manifests itself in a sudden manner, and without being announced by any precursory sign. In some cases, however, the patients experience the symptoms which usually precede gastric fevers, such as loss of appetite, headache, lassitude, watchfulness, prostration, low spirits, &c."¹ Blair states that it is sometimes preceded by a sensation of malaise of several days' duration; but that in one-half of the cases the disease commences suddenly (pp. 64, 72). Savarésy (p. 267) enumerates increased heat, vertigo, slight headache, weakness of the lower extremities—which feel as if cut; hence the denomination *coup de barre*, applied to the disease by Du Tertre and his contemporaries. The greater number of writers on the fever in question² enumerate much the same symptoms, together with faintness, weariness, yawning, pain in different parts of the body, bitter taste in the mouth, yellowish tinge of the tongue, thirst, hard and wiry or full and developed pulse, hot breath, proclivity to the use of strong drinks, costiveness, and diarrhoea. Humboldt states that, in Mexico, the sudden drying of the soap applied to the face for the operation of shaving is regarded by some as a premonitory sign of the disease (p. 774). Other signs might be gathered; but the above will suffice to show the identity of the fever of tropical climates with that of our own country, while the parallel is rendered still closer by the fact, that most of the writers to whom I have referred, state that the disease often, if not generally, attacks in a sudden manner.³

To this we may add, that a difference in regard to the premonitory symptoms will generally be found to depend on the degree of violence and malignancy, as well as on the rapid course of the case. The mild forms of the disease are more frequently preceded by some one or another of the phenomena mentioned, while those of a different character are sudden in their onset.⁴

B. Mode of Invasion.—Passing from the premonitory symptoms to the mode of invasion of the disease, we shall find that it varies somewhat in the several forms assumed by the latter in different subjects and in different epidemic seasons. Dr. Rush states that, in 1793, many went to bed in good health and awoke in the night with a chilly fit. Others were attacked in the day, and in most of them the disease came on with a chill, which afforded, by its violence or duration, a tolerable presage of the issue of the case

¹ Diction. des Sci. Méd., xv. 334.

² Lefoulon, p. 370; Vatable, p. 345; Arnold, p. 8; Copland, iii. 141; Vincent, p. 23; Heastie, p. 18; Mabit, p. 10; Joubert, p. 966; Carter, p. 3; Anderson, p. 3; Pugnet, p. 351; Holliday, p. 8; Gillespie, p. 37; Rochoux, T. A., p. 83; Y. F., p. 263; Jackson, i. 61–65, 86, 102, 107, 140; Rufz, pp. 11, 53; McArthur, p. 345; Dickson, *Fev. of Mariegalante*, p. 364; Levacher, p. 70; Osgood, p. 9; Ralph, ii. 65; Caillot, p. 16; Bancroft, p. 9; Moseley, p. 435; Hillary, p. 147; Hunter, p. 63.

³ Dancer, p. 83; Dariste, p. 158; Lemprière, ii. 59; Gilbert, p. 65; Bally, p. 208; Warren, p. 9; Towne, p. 20; Clarke, pp. 6, 7; Chisholm, i. 148; *ib.*, Manuel, p. 175; Imray, pp. 53, 70; Vatable, pp. 344–5.

⁴ S. Jackson, p. 50; Robert Jackson, i. 61; Ralph, p. 65.

(iii. 52). Dr. Nassy, in his account of the same epidemic (p. 21), states that the disease began with pains in the loins and head, accompanied with chills more or less considerable. Barnwell (p. 369) speaks of a sense of uneasiness and sickness at the stomach, followed by *rigors*, and a stricture all over the extreme vessels, with a shrivelled appearance of the skin. Much to the same purpose are the statements of Cathrall and Currie. The former (p. 21) speaks of some degree of general lassitude, loss of appetite, disagreeable taste in the mouth, sudden prostration of strength, universal pain and soreness, some degree of chilliness, or alternate chills and flushes of heat; but very seldom a complete rigor. Dr. Currie (p. 20) remarks that, after the premonitory signs, which lasted six or eight hours, and sometimes without, "a chilly fit, alternating with glowing flushes of heat, came on generally in the after part of the night, or forepart of the day, which, after a duration for the most part short, though sometimes of twelve or fourteen hours' continuance, gave place to fever." Thus it would appear that in the fever of the year in question the attack was usually ushered in by a chill or chilly fit. It is true that, from Devcze's statement, we might infer that they were only of occasional occurrence and transient character—*quelques frissons rapides et inégaux précédaient parfois une chaleur violente.*" But the statement of this writer, however entitled to our regard, cannot overthrow those to which I have referred; and, after all, shows that chills, if not as frequent as may be inferred from the latter, were nevertheless encountered in many cases.

But, however this may have been in '93, chills do not appear to have been frequently observed the next year. In the epidemic of that season, as we learn from Dr. Rush, "a slight shivering ushered in the fever in several instances. But the worst cases which he saw came on without a chilly fit, or the least sense of coldness in any part of the body" (iii. 214). Three years after, in 1797, the disease in like manner came on often, according to the same writer, without chills (iv. 13); or, as we learn from Dr. Currie, was ushered in with chilliness, or sense of universal coldness, but no shivering.¹ At a later period (1805), there was a chilliness, with sometimes some degree of tremor; generally, however, the latter phenomenon was wanting, and the chilliness was often compared to streams of cold water running along the spine.² In 1820, as we learn from the chronicler of the epidemic of that year, Dr. Jackson, "chills, with few exceptions, occurred at the beginning of the attack, and were frequently distinct rigors; but sometimes amounted only to a sense of coldness, or a crawling sensation. In some cases, and those of the greatest violence, they were entirely absent (pp. 51, 59, 61).

From the foregoing statements of the occurrences in the various periods of the prevalence of yellow fever in Philadelphia, it follows that the disease was ushered in by a chill of more or less duration and severity; that this mode of invasion was of more frequent occurrence during some seasons than others; that the chill was often replaced by a mere sense of coldness; and that in some cases—at certain periods very generally—there was a complete absence

¹ Bil. Fev., p. 18.

² Caldwell, p. 81.

of both the one and the other. To those who are aware of the above analogy of the fever of our city with that of other sections of North America, I need scarcely say that similar observations have there been made. For while by some writers¹ the febrile excitement is said to have been preceded, either generally or in many cases, by chills of greater or less severity and duration; while others—Bayley (pp. 72, 73), Hogg (*Fever of 1839*, pp. 416, 417), Cartwright (ix. 11)—have represented the disease as being ushered in by a mere sense of coldness, sometimes slight, at others deep, penetrating, and stupefying, or alternating with flushes of heat²—there are not wanting those who contend that the disease is but seldom preceded by symptoms of the kind. Some do not allude to them; others, again, think they are as often absent as present. In 232 cases examined at the Roper's Hospital, Charleston, in 1854, with a view to ascertain how often chill was the incipient symptom, it was ascertained that 104 commenced with it, and 128 without.³

Dr. Rochoux, whose observations relative to the yellow fever of temperate climates were made at Barcelona, states that the fever always commenced with a sensation of cold, more or less marked, deep, penetrating, and stupefying, and in some cases of twelve hours' duration; and that in a few instances it came on with a decided chill (pp. 455, 568). Similar, or nearly similar, statements are made by Audouard (p. 55), Arejula (p. 170, and *Edinb. Journ.*, i. 448), Fellowes (p. 61), Berthe (p. 84), Caisergues (p. 167), Palloni (p. 4, and *Edinb. Journ.*, ii. 84), Boyd (*Johnson*, p. 297), Dufour (*Marseilles Journ.*, iv. 50), Proudfoot (*Edinb. Journ.*, xxvii. 249), Jourdain (*Ann. de la Méd. Phys.*, v. 257, 258). On the other hand, we may infer from the statements of other writers that chills were at different seasons of more frequent occurrence than would seem to have been noticed by the afore-mentioned writers. O'Halloran states that, in the inflammatory species, there was a sense of cold at the extremities, gradually extending to the back and limbs, and succeeded by chilliness of long duration. In the serous temperaments, chills were slight and of short duration (p. 123). Dr. Jackson states that in the fever of Cadiz and Xeres, in 1820, alternate chills and flushings of heat were generally noticed—the chills seldom strong, but recurring at intervals for a length of time (p. 94). Pariset (p. 380) mentions horripilations or chills as ushering in the disease. In the two descriptions of the disease which we find in the large but *flat* quarto of the last writer—the one of the fever of Seville in 1819, furnished by Dr. Velasquez, and the other of the epidemic of Cadiz in the same year, for which he was indebted to a physican of that city (probably

¹ Gros, p. 9; Thomas, p. 11; Merrill, ii. 233; Dickson, iii. 253; Irvine, p. 27; Warren, in Tytler, p. 501; A. Hosack, p. 11; Seaman, p. 95; Revere, iii. 224; Munson, p. 179; Smith, Am. J., xxv. 501; Kelly, pp. 375, 377; S. Brown, pp. 11, 12; Drake, xxi. 133; Waring, p. 45; Lining, p. 412; Archer, v. 66; Ticknor, iii. 222; Hill, v. 89; New Orleans in 1819, p. 8; Girardin, p. 33; Valentin, p. 164; Hogg, p. 411; Townsend, p. 143; Baxter, p. 37; Drysdale, i. 37; Nott, Charleston Journ., iii. 9.

² Hosack's Practice, p. 388; Perlee, iii. 11; Dickson, p. 348; H. S. Smith, pp. 115, 125; Potter, Notes to Gregory, i. 13; Dalmas, p. 6; Drysdale, i. 137; Kelly, pp. 375-7.

³ Wragg, Charleston Journ., x. 71.

Dr. Flores)—the onset of the attack is represented as having been sudden, and characterized by chills (pp. 9, 22). By Louis we are told that at Gibraltar, in 1828, fatal cases came on usually with an intense headache, accompanied by *chills*, *shivering*, pain in the limbs, and soon after pain in the back (p. 167). In mild cases, most commonly, at the commencement there were headache, *chills*, followed by a slight degree of heat (p. 175).

If to this it is added that in not a few instances the attack is ushered in by neither chill nor sensation of coldness, the identity of the fever of Europe with that of other parts of temperate regions, so far as regards the mode of invasion, will be placed beyond the possibility of doubt. While agreeing on this subject, and admitting that the yellow fever of temperate regions is preceded by a sensation of coldness or a chill, Dr. Rochoux states that the fever of tropical climates always commences by the development of more or less heat, which soon becomes intense; that few or none experience a sensation of coldness, unless they have been wet and have exposed themselves to night air; and that, even under such circumstances, the sensation is slight and of short duration. Relying on the reality of this difference, Dr. R. derives from it an argument in favour of the dissimilar character of the two diseases. But on this point, as on every other connected with this subject, he will be found at variance with well-attested facts. Already we have seen that, in temperate regions, the fever is far from being always ushered in by a chill or a sensation of coldness; that, in fact, these are very frequently absent, or that, in other instances, they are slight and of short duration. In these respects, therefore, we find that some cases, at least, approximate to the fever of tropical climates, supposing the statement of Dr. Rochoux, as regards the latter disease, to be correct. The question, then, to be ascertained is—not whether in some or many cases the disease commences at once by the development of febrile heat, and without the occurrence of a chill or a sensation of coldness, but whether such is the case to the extent mentioned—whether, in fact, the absence of the latter phenomenon takes place in all cases, and constitutes a characteristic feature of the disease. Now, that on this point Rochoux may appeal for support to high professional authorities is evident. Savarésy, in his minute detail of the symptoms of the fever of Martinique (p. 269), makes no mention of chills, nor, indeed, of a sensation of coldness. Neither does Davidson (viii. pp. 248, 249), who states that the Causos and the yellow fever commence with vascular excitement. Dickinson says nothing of chills, but mentions slight chilliness, which, he says, evidently differs from the chill of other fevers (pp. 125, 169). Other testimony to the same effect might be collected.¹

But while such are the statements of these writers, others of a different kind are more frequently met with; for we find, on reference to the highest authorities, that the opening symptom of the disease in the West Indies and other tropical regions is not very dissimilar to that observed elsewhere. Dr. John Hunter tells us that “there is uneasiness, with languor, followed by

¹ Vatable, p. 345; Imray, Edinb. Journ., liii. 78; Desportes, i. 193; Lempriere, ii. 59.

a sense of chilliness or cold shivering, which are soon succeeded by great heat" (p. 20). "In this disease," says Dr. Osgood (p. 9), "first a lassitude is felt, sometimes with alternate chills and flushes of heat, but at other times with great heat and no chills." According to Hillary, the "disease opens with a small chilliness and horror, very rarely with a rigor, which is soon followed by violent heat and high fever." Chisholm remarks that the patient suddenly becomes giddy, and remains in that state half an hour or upwards (i. 147). "During this paroxysm, the body feels cold, and is overspread with cold sweat." (Vol. i. pp. 148, 149.) In another place he says: "The disease, too, in a few cases, has seized the patient in the manner most other fevers come on, that is, with shivering and a sense of cold." (Vol. i. p. 150.) Bancroft (p. 9) states that when the yellow fever is epidemical, and attacks young and robust men, a slight sensation of cold is among the early symptoms. Lempriere (ii. 59) remarks that sometimes a slight rigor precedes the attack. Dr. Th. Clark, in speaking of the fever as it appeared at Barbadoes in 1793, says: "The disease came on with excessive debility, cold, shiverings, &c.; in short, with the same symptoms as fevers of this country" (p. 99). Dr. R. Jackson speaks of the same symptoms in his description of the several forms of that disease as it is observed in the West Indies and on the coast of Dutch Guiana. Thus, in the mild form of continued fever as it affects the sanguine temperament: "The actual invasion is usually indicated by a sense of cold at the extremities and in the back, a creeping in the flesh, termed horror, sometimes by shivering, and even by shaking (i. 61). In the aggravated form there is more or less horror and shivering. The sensations of cold recur at intervals, sometimes continue long, but are rarely high in degree" (i. 65). In the mild form of fever, ranked in the retrograde series, and engrafted on the gangrenous temperament, "the commencement, like that of most other fevers, is marked by a sense of cold and chilliness, seldom strong, but often of long continuance." (Vol. i. p. 87.) In the slight degree, in phlegmatic temperaments, "a sense of long-continued cold, rather than horror and shivering." (Vol. i. p. 104.) In the concentrated form, "the sensations of cold, so common at the commencement of fevers, are here somewhat peculiar—disagreeable, deep-seated, and in a manner stationary, seldom intermixed with flushings of heat." (Vol. i. p. 108.) Speaking of the second form of the disease, Ralph says: "After some days of slight indisposition, fever would declare itself by the recurrence of severe rigor or protracted sensation of chilliness" (p. 65). And Rufz says of the fever of Martinique, in 1838: "In three-fourths of the cases, the disease was ushered in by a single and regular chill—whether the disease came on abruptly or whether it was preceded by premonitory symptoms. The chill was not of long duration." (*Report*, p. 11.) Barrington (xii. 311), Gillespie (p. 38), Moseley (p. 435), McArthur (in *Johnson*, p. 345), Hume (p. 195), Madrid (p. 30), Furlong (p. 290), Stevens (p. 218), Bruce (p. 186), state that this disease is generally ushered in by chilliness, a sensation of cold, horror, shivering—sometimes of long, though generally of short duration; sometimes of a mild, at others of

a severe and penetrating degree. While a large number of writers,¹ besides those mentioned, represent chills—of greater or less force and duration—and even rigors as of frequent, though not constant, occurrence at the outset of the disease. In fact, if we examine the records of the fever of tropical regions, we shall find the most ample confirmation of the statements here made. It matters not if by some, or many, the occurrence of chills or sensation of cold is denied or passed by unnoticed. Such symptoms may, or possibly really do, constitute exceptions of a rule; but that they do present themselves, cannot, from what precedes, be called in question.

Such being the case, the assertion of Rochoux as to the invariable absence of chills or sense of cold at the outset of the fever of tropical regions, and with it the distinction he wishes to establish on that score between that fever and its namesake of temperate regions, fall to the ground. That in the latter regions those symptoms are more usually noticed, and, when so noticed, are in general more intense than in hot latitudes, may, for what I know, be true; but, admitting such to be the fact, it would be improper to conclude from this difference that the diseases are intrinsically dissimilar; for, on the same ground, we should be justified in pronouncing that the fevers of the various West Indian Islands are different from each other; or, again, that the fever which prevails in a given island at one period is not the same as that which appears there at another season; or even that in the same place and at the same time the cases that prevail, though presenting the same characteristic symptoms, differ essentially; since the disease is not in all found to be ushered in in the same manner. Thus, Hillary, who practised at Barbadoes, Drs. Osgood and Holliday, whose experience was acquired at the Havana, and Chisholm, who encountered the disease in Grenada, appear to have observed those symptoms more frequently and in a more marked manner than was done by Dr. Lempriere and Dr. Baneroff, who practised in Jamaica, or Dr. Imray, who practised in Antigua, or Saverésy and Davidson, who resided at Martinique. While the last-named physicians take no notice of chills, or a sense of cold in the fever of that island, Dr. Catel and Dr. Rufz represent them as occurring in a large number of cases. In Jamaica, those who followed Hunter, saw less of those symptoms than appears to have been the case with that eminent physician. Nay, more, the symptoms in question vary in the same season—appearing pretty constantly in some forms of the disease, and being absent in others (*Ralph*, pp. 65, 671); and if this diversity as to their appearance can occur without leading to a disbelief in the identical character of the disease—if with us they may appear some years in a large number of cases, and be absent at other times, though the disease remain the same under all circumstances, the same diversity may certainly be sup-

¹ Lefoulon, p. 370; Copland, i. 139; Leblond, p. 112; Arnold, p. 8; Blair, p. 64; Joubert, p. 966; Hume, p. 197; Bourdon, p. 9; Mabit, p. 10; Vincent, p. 23; Fever of Cayenne, p. 166; Frost, xiii. 29; Peixoto, i. 412; Pugnet, p. 353; Caillot, p. 16; Bally, p. 209; Maher, p. 848; Catel, p. 11; Dariste, p. 158; J. Clark, pp. 6, 17; Holliday, p. 8; Blanc on Seamen, p. 405; Fontana, p. 72; Pym, pp. 228, 233; Gilbert, p. 65.

posed to exist between the fever of the two regions, even supposing it constant, without lending support to the theory of Dr. Rochoux.

To this let it be added that, in some cases, the disease is ushered in by giddiness, bearing great analogy to that produced by inebriating liquors. Dr. Rush speaks of cases in which it came on while the individual attacked was walking in the street, and imparted the idea that he was drunk (iii. 63). The same phenomenon was noted by other writers.¹ In other cases, it has been known to be ushered in with convulsions (*McLean*, p. 92), syncope, &c.²

It remains to be mentioned, in connection with this subject, that the attack very frequently, if not usually, occurs at night, or rather towards morning, when the patient, after going to bed without feeling much, if at all indisposed, awakens with a chill or cold feeling, or again with decided fever.³ Dr. Blair, who has paid particular attention to this point, remarks that, in Demerara, the hours of 6 A. M. and 6 P. M., when in that latitude the most violent disturbance of the day occurs, were most favourable to the seizure. In 1,145 cases, 237 occurred at the first of those periods, and 124 at the other. Fifty-seven took place at midnight. Dr. J. Davy remarks, in relation to this subject, that at the time of sunrise—the coolest period of the twenty-four hours, when, according to the above observations of Dr. Blair, a large proportion of attacks commences—the temperature of the body is lowest. This is the result of a series of trials conducted on his own person consecutively for three years in the West Indies; at the same time the pulse was commonly slowest. This condition of the system might, as Dr. Davy very properly thinks, explain the occurrence in question.⁴

CHAPTER V.

CIRCULATORY SYSTEM.

IN treating of the symptoms of the yellow fever, as they manifest themselves in the circulatory system, I shall examine them under different heads: 1st, I shall inquire into the appearance and character of the blood during the various stages of the disease; 2d, I shall treat of the action of the heart and arteries; 3d, of the capillary circulation and of the hemorrhagic tendency.

A. BLOOD.—From an early period in the history of the disease in this city, the peculiar appearance and character of the blood, both in an early and an

¹ Jackson, p. 358; Cartwright, ix. 13; Merrill, ix. 245; Washington, Med. and Phys. J., vi. 311; Chisholm, i. 148; Bruce, p. 186; Hillary, p. 147; Moseley, p. 435; R. Jackson, p. 88; Frost, xiii. 29; Osgood, p. 11; Pariset, p. 401.

² Bally, p. 228; Rush, iii. 209; Arnold, pp. 7, 8; Gilbert, p. 66; Deveze, p. 26; Valentin, pp. 173, 4; Fellowes, pp. 52, 54; Gillespie, p. 41.

³ Arejula, p. 170; Copland, iii. 141; Rush, iii. 52; Cathrall, p. 21; Thomas, p. 37 (2d ed.).

⁴ Blair, pp. 72-3, and note.

advanced stage, were made a subject of special inquiry. In his account of the epidemic of 1793, Dr. Rush noticed, 1st. That in the greatest number of cases it was without any separation into crassamentum and serum, and of a scarlet colour. 2d. In many cases, there was a separation of the blood into crassamentum and *yellow* serum. 3d. In a few cases, in which this separation took place, the serum was of a *natural* colour. 4th. In many cases, the blood was as sizzly as in pneumonia and rheumatism. 5th. In some instances, it was covered above with a blue pellicle of sizzly lymph, while the part which lay at the bottom of the bowl was dissolved. In two cases, the lymph was mixed with green streaks. 6th. It was, in a few instances, of a dark colour, and as fluid as molasses. To this Dr. R. adds, that, "in some patients the blood, in the course of the disease, exhibited nearly all the appearances which have been mentioned. They varied according to the time in which the blood was drawn, and the nature and force of the remedies which had been used (iii. 142-3). In the year 1794, the blood exhibited the following appearances: 1st. It was dissolved in a few instances. 2. The crassamentum of the blood was so partially dissolved in the serum as to produce an appearance in the latter resembling the washings of flesh in water. 3d. The serum was so lightly tinged of a red colour as to be perfectly transparent. 4th. The serum was, in many cases, of a deep yellow colour. 5th. There was, in every case in which the blood was not dissolved, or in which the second appearance that has been mentioned did not take place, a beautiful scarlet-coloured sediment in the bottom of the bowl, forming lines or a large circle. It seemed to be a tendency of the blood to dissolution." 6th. The crassamentum generally floated in the serum, but it sometimes sank to the bottom of the bowl. In the latter case, the serum had a muddy appearance. 7th. In very few cases there was no separation of the crassamentum and serum. It was of a deep scarlet. In 1793, this appearance was very common. 8th. In others there was partial separation, and the blood was of a deep black colour. 9th. In several instances, there was a transparent, jelly-like pellicle, which covered the crassamentum, and which was easily separated from it without attaining its texture. 10th. The blood, towards the crisis of the fever in many people, exhibited the usual forms of inflammatory crust. It was cupped in many instances. 11th. When the blood happened to flow along the external part of the arm, in falling into the bowl, it was so warm as to excite an unpleasant sensation of heat in several patients (iii. 222-3). In 1797, "the blood was dissolved in a few cases. That appearance of the fluid which has been compared to the washing of flesh, was very common (iv. 12). It was more or less sizzly towards the close of the disease in most cases."

Kindred statements are made by other writers as regards the state of the blood in the early epidemics of this city, though principally in reference to its appearance after death. Drs. Physick and Cathrall found the blood in the heart in a fluid state, and similar to that of persons who have been hung or struck by lightning.¹ Dr. Deveze states that the right auricle was distended

¹ Rush, iii. 92; Cathrall, Fever of 1793, p. 70.

with fluid black blood, or coagula of the same colour.¹ Nassy speaks of the fluidity of the blood.² "The first blood drawn in the fever," says Dr. Currie, "seldom showed any white, glutinous covering, or size; but, after the second bleeding, this appearance was seldom absent, though it was never thick or very tough. The crassamentum was bulky, flat, and smooth, and never rose with that cup-like, or sand-box form, which is common in cases of pneumonic inflammation."³ Dr. Cathrall, already cited, remarks, in his treatise on the same epidemic, that the blood drawn in the early stage "very seldom separated into serum and crassamentum; when it did, the former was nearly of a natural colour, though small in quantity in proportion to the latter; the crassamentum was generally of a florid, arterial colour, tender in the course of the warm weather; as the cool weather advanced, and the disease became more inflammatory, it was sized in some cases, with a proportionable degree of contraction of its superior circumference exhibiting a cup-like appearance. This was generally in proportion to the density of the crassamentum and firmness of the buffy substance, though sometimes during the warm weather there was a thick, tender, sized coat on its upper surface, without the cup, or, as it is generally called, salt-box appearance. When blood was drawn during the yellow suffusion of the skin, the serum was of a corresponding colour, but not in the least bitter to the taste, that could be observed in a variety of cases, and, like healthy serum, it was affected by heat and acids" (pp. 47-8). By all, the fluid condition, and the discoloured appearance of the blood when issuing from the natural outlets of the system in the closing stages of the disease, were noticed.

Much the same remarks were made in respect to the state of the blood, during life and after death, in the epidemics which subsequently prevailed in this city.⁴ In that of 1805, Dr. Caldwell found not only that the blood which flowed in hemorrhage was never capable of firm coagulation, thereby evidencing "that its vitality was nearly extinguished (pp. 85-7), and that in many cases it was dissolved (p. 87); but that, when drawn from the vein, it was seldom covered with a buffy coat; and that the venous blood was very florid, and did not coagulate firmly (p. 104). Similar observations, as to the fluidity, defective coagulation, and dark colour of the blood, were made during the fever of 1820. But, on this occasion, it was discovered that, although the blood presented this appearance, and, when examined after death, was found in a fluid state, its power of coagulation was often rather tardily established than lost. Thus, as we are informed by Dr. Jackson, "the veins of the omentum mesentery, and, in fact, the whole system of the vena portæ, were always distended with fluid blood. It was at first supposed that the blood, being thus fluid, was in a dissolved state, so often mentioned by writers. But Dr. Hewson, wishing to make some experiments, collected portions of it in cups. In the course of ten or fifteen minutes it was firmly coagulated, and this was found, in subsequent observations, invariably to occur (p. 80).

¹ *Traité de la F. J.*, p. 60.

² *Obs. on the Nature Causes, &c.*, p. 28.

³ *Fever of 1793*, p. 41, note.

⁴ *Firth*, pp. 35-6; *Louber Med. Mus.*, v. 23.

From all that precedes, it results that, in the epidemics mentioned, the blood, though in some cases exhibiting, at a particular period of the attack, some at least of the characters it usually assumes in inflammatory fevers, and in diseases based on the existence of local irritation, was more frequently found in an altered and morbid state, as evinced by its often not separating into serum and crassamentum; by the yellow colour sometimes assumed by the serum; by its being sometimes from the beginning, though more generally in the advanced stage, thin, fluid, or thick as molasses, or dissolved; by its occasionally bearing some resemblance to the washing of flesh in water; and, in other instances, by its manifesting considerable tardiness in coagulating; by its very generally coagulating imperfectly, and sometimes by its *not* coagulating at all. It remains to state, in reference to the occurrences in this city, that the appearance of the blood during the epidemic of 1853, and in the cases that presented themselves this year (1854), most of which fell under my personal observation, differed in nothing from those noticed in preceding seasons. In few, was there a show of inflammatory crust; in the greater number, the crassamentum, when formed, was large, flat, soft, and easily broken; in some, the separation did not take place; in many, the colour was darker than in the natural state; the serum in several was yellow; and in many instances, in less proportion to the solid parts than in health and other complaints; while, in all cases, as the disease advanced, the blood became thin, black, and otherwise morbidly altered.

A diseased condition of the blood, similar to the one noticed in the yellow fever of this city, had long before been recorded, with more or less minuteness, by writers in this and other countries. Lining states that, in the fever of Charleston, "the blood saved at venesection had not any inflammatory crust. In warm weather it was florid, like arterial blood, and continued in one soft homogeneous mass, without any separation of the serum after it was cold. When there was any separation, the crassamentum was of too lax a texture."¹ In a subsequent page, he speaks of a partial dissolution of the blood in the after-stages of the disease, as exhibited in the fluid discharged in various hemorrhages.² Moultrie states that, in the first stage, the blood taken in venesection is dissolved, the crassamentum is broad, thin (*crassamentum ejus latum minimeque profundum*), floating on the surface. It is of less firmness than in the natural state, and exhibits, at times, livid spots on the surface. In the last stage it is black, dissolved, and in a state of putrefaction (p. 7). From that day to this, all writers on the fever of this country, with scarcely one exception, mention incidentally, or call special attention to, the altered state of the blood throughout the whole course of the disease, or during its latter stages: its defective coagulability, the absence of inflammatory crust; the softness of the coagulum; the absence of separation; the peculiar appearance or small proportion of the serum; the dark colour of the whole mass; its altered consistence, &c.³

¹ Edinb. Med. Essays and Obs., ii. 415.

² Ibid., p. 422.

³ S. Brown, p. 15; Valentin, p. 182; Irvine, p. 25; Miller, p. 62; Mitchell, Med. Mus., i. 6, 7; Kelly, Am. J. (N. S.), xiv. 389; Harrison, N. O. J., ii. 139; Stone (F. of Natchez),

If we turn now to the yellow fever of tropical regions, we shall find that matters do not take a different turn; for, though the condition of the blood is not attended to by Du Tertre, Labat, Lignon, and others of the old chroniclers of the disease, it did not escape the attention of, and is dwelt upon with more or less emphasis by, the early writers on the subject, and has never ceased since to be mentioned as one of the principal characteristics of the fever. Thus, Dr. Towne, who practised in Barbadoes in the early part of the last century, and was, I think, the first English writer on the yellow fever, speaks of the "too great division of the constituent parts of the blood" in the early stage, and remarks that, in the after part of the disease, it "becomes a lazy, stagnating, inactive puddle."¹ Warren, who came some twenty years after, states that the blood drawn in the hot stage "is florid and rarefied, with evident marks of colliquation, and without the least sign of a size that I could even once observe; the *insula* or *crassamentum* (even when cool), upon moving it, undulates like water in a basin, and has sometimes blackish spots here and there upon its surface, with much yellow serum, upon which it extends itself wide and thin." Hume, in like manner, noted the broken-down and dissolved texture of the blood, particularly in the latter stages (p. 199). So, also, Makittrick, who says: "The blood is apparently firm, but, when exposed to the air, it assumes less consistence than in the natural state. The crassamentum, on the third day, becomes of a blackish colour, and is scarcely coagulated. Hence, it is easily decomposed, or it easily becomes putrid." Not very different is the account given by Nasmyth and Bruce: "The blood, in the beginning," as the former remarks, "is commonly loose and dissolved;" a condition to which he attributes the passive hemorrhages which take place at some period of the disease.² The other says of the blood in the first stage: "Sanguis etiam ingruenti morbo missus, colorem exhibit floridum, rutilum, et quasi rarefactum, crassamento vix cohærente, sero luteo croceo"—and in regard to the stage of prostration, he remarks that the blood which passes off is almost black.³ "The blood obtained by venesection," says Desportes,⁴ "is always very red, florid (vermeil) and frothy, containing but little serum. If we bleed, after the cessation of the fever, the blood remains a long time in a liquid state, sometimes three or four hours after the operation, and there is no serum." Hillary is equally explicit: "Blood taken from the patient, even at the beginning of the disease, is often of an exceeding red colour, much rarefied and thin, and without the least appearance of siziness; and the crassamentum, when it has stood till it is cold, will scarce cohere, but fluctuates; the serum is very yellow."⁵ "Blood taken from the sick on the second or third day, is much more

N. O. J., vi. 571; *Ib.*, N. O. J., ii. 181; Cartwright, *Med. Rec.*, ix. 26-7; Drysdale, *Med. Mus.*, i. 138; Gros, *F. of N. O.* in 1817, p. 11; Dalmas, p. 16; E. H. Smith, p. 122; Archer, *Med. Rec.*, v. 68; Nott, *Am. J., N. S.*, ix. 281; Townsend, p. 196; Cooke, *N. O. J.*, x. 621; Lewis, *ib.*, i. 301.

¹ A Treatise of the Diseases, &c., in Barbadoes, pp. 21, 54.

² Lind on Seamen, p. 55, *note*.

⁴ *Maladies de St. Domingue*, i. 215.

³ Lind on Hot Climates, pp. 278, 9, *note*.

⁵ *Change of Air, &c.*, p. 147.

dissolved, the serum more yellow, and the crassamentum florid, loose, scarce cohering, but undulates like sily water when shaken, and sometimes has dark blackish spots on its surface, showing a strong gangrenescent diathesis."¹ In the last stage, the "blood is so attenuated and dissolved as to flow from different parts."²

Another writer, who also observed the disease in the West Indies, says, that the crisis of the blood "is as much broken down before death, and its vitality destroyed, as it could be by the introduction of the poison of the serpent's tooth;" and that "after death it will be seen to have lost all the character and composition of blood, being found in its vessels like the lees of port wine, or the grounds of coffee."³

Dr. Stevens dwells minutely on the state of the blood in the various forms of West Indian fevers, and in an account which, though too highly coloured, embodies many true facts, states, as the result of his observations, that in that form which he denominates African typhus, and which corresponds to our yellow fever, the blood is diseased even before the attack. When drawn in the beginning, it is dark in colour, and decidedly deranged. It coagulates almost invariably without any crust. Black spots are seen on the surface of the crassamentum. The coagulum is easily broken up with the finger, and during its formation a large quantity of the black colouring matter falls to the bottom of the cup. When the serum separates it has a yellow, even a deep orange colour (p. 219). In the first stage, the structure of the red globules is frequently deranged, as evinced by the fact that the colouring matter is often detached from the globules, and dissolved in the serum. This gives to that part of the blood, when separated from the fibrin, a bright scarlet colour, which cannot be separated from it. As the disease advances, the red colour is lost, and the whole becomes black, and so thin as no longer to resemble healthy blood.⁴ The change of colour is not limited to venous blood, but extends to the fluid circulating in the arteries; the whole changing from a natural scarlet, or modena red, to a dark black, having great analogy to the matter of black vomit. The blood found after death has the character of a dissolved fluid, nearly as thin as water, almost as black as ink, and evidently so diseased as to be totally incapable of either stimulating the heart or supporting life. In both cavities of the heart the fluid is equally black, and, in the whole vascular system, all distinction between arterial and venous blood is entirely lost.⁵ Of the blood, as he observed it at Barcelona in 1821, Pariset remarks: "The blood found in the cavities of the heart is black and fluid. If clots exist, they are small and diffuent." In another place, he speaks of the blood as being apparently deprived of fibrin, and no longer red; as no longer separating itself into a solid part and yellowish serum, and as always remaining black, carbonized, and fluid. "The blood," he adds further on, "is certainly more fluid and blacker. The arterial would seem to approximate to the

¹ Op. cit., p. 148.

³ Ferguson, Recol., p. 146.

⁵ Ibid., pp. 2, 3.

² Ibid., p. 151.

⁴ Observations, pp. 4, 5.

venous, so far as regards colour and consistence. Its molecules lose, in great measure at least, their property of cohesion—their plastic form” (pp. 361, 363, 429).

Enough, I have no doubt—perhaps too much—has already been said to show that in the yellow fever of this and other countries, the blood, from the onset of the disease, has been found variously, but certainly deranged; and that this morbid change becomes more and more evident as the case progresses. Nor is it alone on the testimony of the foregoing writers that such a condition of the blood can be established. So far from this being the case, there is scarcely an author who has treated of the disease—whether in tropical or temperate regions—whatever may be his pathological views, but has noted some or many of the changes in question in the early or later stages, or after the death of the individual attacked.¹

From a survey of all that precedes on the subject, and of various statements contained in the writings of other medical inquirers, as well as from facts which have come under the observation of the author of these pages, we arrive at the following conclusions:—

1. In some instances, the blood appears at first unaltered as regards colour, proportion, firmness of the crassamentum, power of coagulation, quality of the serum, &c., and continues so till the accession of the second stage.²

2. More generally, and even in cases in which, judging from its general aspect, it would seem to preserve its usual characters, the blood is found more or less—often very considerably and as often completely—deprived of its power of coagulation; the crassamentum, when the separation takes place, being generally found broad, flat, thin, of soft consistence, and diffuent.³

3. But this inability or reluctance to coagulation, though generally exhibited, is not universal, and cases occur in which the blood coagulates with as much readiness and firmness as in other diseases. Dr. Kelly remarks, that he has

¹ Dariste, pp. 125–127; Copland, iii. 141; Boyle, pp. 114, 167; Gillespie, pp. 42, 72; R. Jackson, i. 294, &c.; Rouppe, p. 307; Ib., Transl., p. 411; Holliday, pp. 10, 11; Arnold, pp. 24, 35; Hunter, pp. 64, 124, 155; McLean (H.), p. 32; Blair, p. 84; Hume, pp. 198–9; Levacher, pp. 76, 82; Heastie, p. 21; Frost, xii. 30; Joubert, p. 971; Savarésy, p. 273; Fever of Cayenne in 1850, p. 176; Lempriere, ii. 273; Magne An. Marit. 1844, iv. 552; Maher, p. 887; Ralph, ii. p. 77; Vincent, p. 45; Mabit, pp. 11, 12; Catel, p. 15; Evans, p. 226; J. Davy, Ed. J., lxxii. 280; Rochoux, F. J., p. 157; Imray, liii. 91; Wilson, p. 24; Williamson, i. 127; Nichols, in Johnson, p. 102; Rufz, p. 15; Pugnet, p. 357; Bally, p. 202; Chirac, i. 57; Louis, p. 75; Doughty, pp. 14, 95, 106, 118, 132–4, 160; Gillkrist, ii. 280; Fellowes, pp. 55, 62; Blair, p. 9; Audouard, p. 221; Berthe, pp. 88, 255; O'Halloran, p. 112; Palloni Firmin, *Maladie de Surinam*; Pennell, *Med.-Chir. Trans.*, xxxvi. 253; De Maria, p. 61; Arejula, p. 427; Lallemant, *Observações Acerca da Epidemia*, &c., p. 8, Rio Janeiro, 1851.

² Rush, Rochoux, Catel, Blair.

³ Rush, Caldwell, Cathrall, Lining, Moultrie, Warren, Bruce, Hillary, Stevens, Jackson, S. Brown, Irvine, Kelly, Harrison, Stone, Cartwright, Lempriere, Rufz, Bonc, S. H. Jackson, Miller, Townsends, Palloni, Pariset, Vincent, Mabit, Valentin, Ralph, Drysdale, Savarésy, Nasmyth, Levacher, Hunter, Caisergue, *Dict. des Sci. Méd.*, Holliday, Rouppe, Gillespie, Catel, Evans, Hackett, Gillkrist, Rochoux, Imray, Firmin, Hume, Makittrick, Bryson, Gros, Fellowes, Blair, Audouard, Berthe.

seen the blood coagulate as perfectly in the yellow fever as in ordinary intermittent fever.¹ Dr. Stevens states that in that form of pyrexia which he denominates *elimite fever*, the fibrin coagulates firmly.² Dr. Evans found the blood diseased in only 11 out of 26 fatal cases, and in 12 out of 37 others (p. 225); which, admitting them all to have been genuine yellow fever cases—which I do not—is a small proportion. But be the number small or large, some of those in which the blood did not appear to the reporter to have been diseased, were doubtless of that sort, and necessarily furnish us with examples of firm and natural coagulation in yellow fever. Dr. Frost says: “The blood drawn was in some dense and almost black, and if allowed to stand a little while, was as compact and solid as the substance of the liver.”³ Drysdale states, that “in some cases the crassamentum was very firm.”⁴ The retention of the power of firm coagulation might, in the absence of positive facts, be inferred from the statements already cited in reference to the manifestation of particular conditions of the blood usually associated with the power in question, and to which I shall revert presently. To this I must add, that instances have fallen under my observation in which the blood, in respect to the power in question, differed in no wise from blood in health, or in diseases of a different character.⁵

4. In many cases, the power of coagulation, though not lost, is tardy in manifesting itself; often it does not do so until the blood has been for some time removed from the body and exposed to the action of the atmospheric air. We have seen the statements on the subject of Dr. Jackson, of this city (p. 80), Desportes, in the West Indies, and others. Dr. Doughty drew some blood (two ounces) for experiment. “For two hours there was but little disposition to coagulate; it then began gradually to coalesce in its particles, from the bottom of the pot, and in the course of six hours there was a general appearance of coagulum, without the slightest separation of serum, however, and it was of so slender a texture, that the immersion of the feather end of a quill returned it instantly into one homogeneous mass.” When the patient had recovered, or nearly, blood was again taken for further experiment. This formed, in a few minutes, a very firm coagulum, which could not be separated, as before, by the immersion of a feather. Next morning there was a separation of serum to nearly half the quantity of the whole mass, of a deep green-like yellow colour, very much resembling that of urine (pp. 160, 1, 8). Dr. Nott collected the blood by thrusting a trocar into the right auricle, and drawing it off in clean quinine bottles. It was so fluid, and was accumulated in such quantity in the auricle, and veins connected with it, that ten or twelve hours after death it would run freely through the canula to the amount of a pint or more. When set aside, it generally coagulated; in some cases this was effected in fifteen or twenty minutes, but in others not before several hours—even as late as thirty-six.⁶ The same tar-

¹ Am. Journal of Med. Sci., N. S., xiv. 389.

³ Med. Repos., xii. 30.

⁶ Davidge, Dickinson, Blair.

² Stevens on the Blood, pp. 206-7.

⁴ Med. Mus., i. 138.

⁶ Am. J., ix. (N. S.) 281.

diness has been noticed by Blair (p. 84), S. Brown (p. 15), and Harrison (p. 140).

5. In many instances the blood, from the onset of the attack, though more generally after the accession of the later stages, not only manifests a reluctance to coagulate, but remains fluid, and, as it were, dissolved. We have seen that this peculiar state of blood was noticed by Dr. Rush, Physick, Cathrall, Caldwell, Nassy, and other historians of our early epidemics. It was found in our more recent visitations. It is mentioned by Lining, Moultrie, Hume, Nasmyth, Bruce, Hillary, Hunter, and Stevens; and has been noticed and dwelt upon by other writers in this and other countries—Evans, Frost, Pariset, Stone, Irvine, S. Brown, Miller, Davidge, Cartwright, Caisergues, Ralph, Townsend, Catel, Valentin, Vincent, Mabit, R. Jackson, Imray, Levacher, Gros, Audouard, Berthe, Nott, Blin, Joubert, Bryce, Veitch, Bone, Bryson, S. H. Jackson.¹

6. In some cases during most epidemics, and very generally during others, the blood is found sizy, and even cupped, and presenting, more or less exactly, the appearance described by Dr. Rush. Drysdale, during the epidemic of Baltimore, observed cases in which the crassamentum was covered with a thick buffy coat, cup-formed, and floating in a large quantity of serum. In other cases, in which there was no separation, a quantity of very yellow lymph floated on the top of the red globules till it coagulated—the whole congealed into a solid mass.² Chisholm states that the blood drawn at an early period always threw up an inflammatory crust of greater or less thickness (i. 336). Rochoux says that, in the West Indies, after the first forty-eight hours, the blood in one-half of the cases is sizy. In some instances, the size is a simple jelly of a pearl-gray colour—tremulous, semi-transparent, and from half to one line thick. In other instances, it is of a net-like character, through the openings of which the crassamentum is seen, covered or not with a thin gelatinous pellicle. In others, again, it is from one to three lines thick, and of a yellowish colour.³ Other writers, as Williamson, Dickerson, Cathrall, Currie, Savarésy, Grant, and Catel, speak of a buffy coat.

7. More frequently the blood does not present a buffy character, and is neither sizy nor cupped. We have just seen that this freedom from size was often, if not generally, noticed in our early epidemics. It was common—nay, almost universal—in that of 1853, and among the cases which occurred the next year. According to Rochoux, the appearances in question were absent in one-half the cases which fell under his observation; and few, if any of the writers above referred to speak of them otherwise than as of rare occurrence. In like manner, Drysdale states that in some instances they were absent in Baltimore, in 1794. Dr. Davidge remarks that, in 1798, in the same city, the blood, when abstracted, was seldom covered with a buffy coat (i. 104). “Never,” says Dr. Gillkrest, “has the blood, in a single instance, presented a buffy surface” (ii. 280). Harrison affirms that, in New Orleans, blood

¹ Report on Fever of Cayenne.

² Med. Mus., i. 138.

³ Rochoux, *Traité de la Fièvre Jaune*, i. 157, 158; *Ib.*, *Des. Dif. Mal.*, &c., pp. 339, 341.

drawn from the arm rarely, if ever, presents a buffy coat, and that he has never seen it cupped (p. 140). Rufz (p. 16) made a like observation in Martinique in 1838. Catel (p. 15) speaks of an albuminous coating as only of occasional occurrence. Frost did not recollect to have seen a buffy coat (xiii. 30). Moseley (p. 445) remarks that the surface of the blood is never sizy, and seldom contracted; and similar observations are recorded by Lining, Warren, Hillary, Moultrie, Towne, Hume, Ferguson, Hacket, Stevens, Nott, Trotter, R. Jackson (*Fever of Jamaica*), Bone, Davidge, Bryson, Boyle, Lempriere, and Pennell.

8. In some instances—during some epidemics and seasons, very generally—the blood, at the onset of the disease, is of a florid, brilliant red or scarlet colour. This appearance, which is more common when the circulation is more than usually rapid, was noticed, as already seen, in the epidemics of this city;¹ and such, also, was the case at Grenada in 1793, according to Chisholm (i. 336). A like appearance has been noted by Palloni, Catel, Rouppe, Caisergues, Dickinson, Stevens, Moseley, Wilson, Rochoux, Hillary, Fournier, Lining, Bruce, Warren, Desportes, Stevens, Trotter, Hume, Wilson, and Pennell, as of frequent or occasional occurrence.

9. In all cases, whatever be the appearance of the blood at the onset of the attack, it becomes dark, and even black as the disease advances and passes to the last stage. In many instances, and during some epidemics, in the large majority of cases, it exhibits a dark hue throughout the whole course of the disease; and in all instances it is found so after death. I have already referred to Rush, Physick, Cathrall, Deveze, Moultrie, Bruce, Makittrick, and Stevens, for statements to that effect; and, if we turn to the writings of Savarésy, Williamson, Bruce, Maher, Joubert, Chabert, S. Brown, Stone, Bone, Catel, Bryson, Caisergues, Copland, Levacher, Nichols, Rufz, Dariste, Jackson, Brown, Kelly, Cartwright, Dalmas, Pariset, Vincent, Mabit, Valentin, Evans, Palloni, Drysdale, Miller, E. H. Smith, Gillespie, Frost, Rouppe, Towne, Pugnet, Bally, Hacket, Wilson, Gros, Blin, Fellowes, Audouard, Berthe, Doughty, Blair, and Pennell, we shall find that the change has been noticed everywhere.

10. In some instances, according to Warren, Hillary, Moultrie, Stevens, and Caisergues, the crassamentum presents dark blackish spots on its surface.

11. It is not unfrequent when the blood is dark, while issuing from the body, to find it becoming more florid as it flows, or after exposure to the air. (*Drysdale, Rufz.*)

12. In some instances, the blood presents a mixed appearance, as if the vein had contained two differently-coloured fluids—the one bright red, the other almost black. This was observed by Dr. Imray, at Antigua, in 1838.

13. In some instances, the blood has been found, even at the onset, quite fluid and of thin consistence. Dr. Rush and others noticed this peculiarity in this city, and it is mentioned as of frequent occurrence in several works already

¹ Rush, Cathrall, Caldwell.

referred to—Hillary, Caisergues, Fournier, Nicholls, Townsend, Miller, Nott, Frost, Hacket, Levacher, Imray. This character is more frequently observed in the latter stages, and often only after death.

14. In other cases, it is represented as thicker, in the early and after-stages, than in health, and as having the appearance of molasses. The same character of the blood is noticed after death. (*Evans, Cartwright, Hacket, Bally, Savarésy.*)

15. In some instances, there is an uncommon reluctance in the flow of the blood, although the pulse may feel strong and full. (*Brown, Evans.*)

16. As regards the arterial blood, Dr. Hacket (*Med.-Chir. Rev.*, xvi. 291) found it, in the living subject, to present its characteristic bright-scarlet hue. But that such has not always been the case, is fully proved by the statements of older and modern writers. Towne observed that not only the venous, but the arterial blood also, was dark, and became “a lazy, stagnating, and inactive puddle.” Dr. Stone found it black at Woodville in 1844, and at Natchez four years after. Pariset affirms that the arterial blood presented the same characters as the venous, in regard to colour and consistence, at Barcelona in 1821. We have also seen that similar observations were made by Dr. Stevens, of Santa Cruz; and every dissection, those of Dr. Hacket not excepted, shows that, after death, the blood found in the left cavities of the heart differs in nothing from that contained in the right.

17. Dr. Rush is not the only writer who has noticed the increased temperature of the blood in the early stage of the yellow fever. Dr. Drysdale remarks that, at Baltimore, in 1794, it was sometimes so hot as to impart a scorching sensation to the arm; and Dr. Rufz, of Martinique, called attention to the same peculiarity in his account of the epidemic of 1838, '9 (p. 16). Haller, in the second volume of his great work on Physiology (p. 36), cites authorities to prove that, in yellow fever, the temperature of the blood has been known to rise to 102 degrees; and facts might be adduced to show that it has risen higher still. It has been found that the temperature of the body reaches to from 104 to 110 degrees—that in health being 98½. A corresponding increase in the temperature of the blood may naturally be expected to occur; for, as is remarked by Dr. Babington, whatever theory may be adopted respecting the generation of animal heat, it is a fact, which is generally admitted, that it is effected through the medium of the blood; that it is, *cæteris paribus*, increased in proportion to the velocity, freedom, and force of the circulation; and that it is mainly dependent for its development on the presence of red particles.¹ The increased heat of the blood beyond the healthy standard, in the early stage of some forms of yellow fever, is placed beyond a possibility of doubt, and cannot be matter of astonishment. It is, of course, not peculiar to the disease, and is generally, though not always, found in other fevers and the phlegmasia. In intermittents it has risen to 104; in other fevers, to 106 and 108; and in continued fevers, to 109. (*Haller.*)

¹ Cyclop. of Anat. and Phys., i. 418.

18. It has been seen that while changes are effected in the character and appearance of the blood generally, and in the consistence and colour of the crassamentum, alterations of equal importance and extent are noticed in the serum, both in respect to quantity and coloration.

a. In many instances, the number varying at different seasons, there is no separation into crassamentum and serum, the blood drawn retaining its homogeneity—and this, whether it coagulates or remains fluid. In this city, this character was frequently observed during the epidemic of 1793; less so in 1794. It was likewise observed in Baltimore in 1794 (*Drysdale*, i. 138), and is noticed by Evans, Frost, Lining, Gillkrest, Lefort, and Nasmyth.

b. In many cases, when the separation into crassamentum and serum takes place, the quantity of the latter is much smaller than in the normal state, especially in the early stage of the disease. Dr. Mitchell, of Virginia, found the proportion of the serum to be one-half in the beginning, two-thirds at the height, and larger in the after-part of the disease; and Desportes represents the blood, in the early stage, as containing but little serum. Similar facts were shown here by Cathrall and others; at Martinique by Rufz and Catel, and elsewhere by Williamson, Rouppe, Savarésy, Grant, Audouard, &c.

c. On the other hand, *Drysdale* remarks that in some cases the quantity of serum was in excess (p. 138). Dr. Imray, too, states that at Antigua, in 1838, the serum was in larger quantity than natural, even at the onset of the attack (pp. 91, 92); so, likewise, Gillespie (p. 71) and Moultrie (p. 3).

d. Like Dr. Rush, Dr. *Drysdale* states that in some cases the serum had the appearance of water in which meat had been washed (p. 138). Dr. Caisergues makes similar remarks as to the blood issuing from the outlets of the body (p. 159).

e. The serum is usually found of a yellow or an orange colour. This, as we have seen, was early noticed in this city by Drs. Rush and Cathrall, and has been observed by many others. It is mentioned by Warren, Hillary, Stevens, Bally, Palloni, Caisergues, Stone, Rouppe, Fournier, Gillespie, Pariset, Frost, Potter, Stevens, Archer, S. H. Jackson, *Drysdale*, &c.

f. In some instances, a portion of the red or black extraneous matter is precipitated to the bottom of the cup, or the serum mixes intimately with these, and acquires thereby a bright scarlet, or dark, or muddy hue, as the case may be; all of which have been noticed by Rush, Stevens, Catel, Imray, Potter, and Nott.

19. It remains to remark, in reference to the state of the blood in yellow fever, that it has been found to emit a peculiar smell, which differs from that emitted by the blood in other fevers—plague, typhus, typhoid, &c.—and is probably produced by a volatile salt of ammonia. This fact was noticed in the West Indies by Dr. Stevens (p. 219), in relation to that form of the fever he denominates the African typhus; and Dr. Rush, in his account of the epidemic of 1794, states that a similar fact was communicated to him by a German bleeder, whose experience in 1793 was very ample, and affirmed he could distinguish the yellow from all other forms of fever. “From the cer-

tainty of his decision in one case which came under my notice," adds Dr. R., "before a suspicion had taken place of the fever being in the city, I am disposed to believe that there is a foundation for his remark." (Vol. iii. p. 223.) I may add that Mr. Ripperger, an experienced bleeder and highly respectable man, who has gone through all our epidemics, from 1793 (inclusive) to the present day, and during that time—thanks to the system of practice long in vogue—shed more blood than any ten men living or dead, has confirmed to me the above statement of Dr. Rush's informant. And why should it not be so? It is known, particularly since the experiments of Barruel, that the blood of every vertebrate animal has in it an odoriferous principle, identical in all the individuals of the same species, and similar to the odour of the cutaneous transpiration, or to that part of it which gives to each animal its characteristic smell. We know also that, according to the principle laid down by Barruel, and more or less acknowledged by other chemists, the blood of each individual exhales an odour closely resembling that of the cutaneous perspiration; and so peculiar, that the species, and even the sex, of any animal, from whom a given quantity of blood has been drawn, may be determined by it. It is known, also, that certain poisonous and other substances—ether, hydrocyanic acid, camphor, and alcohol, which, when taken into the system, find their way into the circulation—impart peculiar odours to the blood; and, when we combine these various circumstances together, we cannot help perceiving that the yellow fever, like other malarial and zymotic poisons, when absorbed, may so modify the fluid as to produce analogous effects.

In the latter stage, towards the close of life, the blood discharged by hemorrhage is represented, by Chisholm, Brown, Caisergues, Palloni, and Berthe, as emitting a highly offensive smell. But, by Dariste and many other writers, the statement has been invalidated; and my own observations lead me to unite with them in opinion, and to ascribe the effect to the changes which the fluid undergoes after exuding from the capillaries.¹

The preceding facts and statements can leave no doubt on our minds that the blood in the yellow fever approximates, to a great extent, to the condition it presents in other pyrexia of the zymotic class. It exists, in varying degrees, in all malarial diseases, from the simple intermittent to the malignant remittent, as well as in typhoid, typhus, and relapsing fever, to say nothing

¹ It would be unsafe to judge of the state of the blood circulating through the system by that of the fluid oozing from the capillaries of the mouth, anus, stomach, ulcers, &c., for facts are adduced to show that, while the latter may appear dissolved, the former may retain many of its physical properties. Dr. Blair gives the case of a man who was bled from the arm to $\frac{3}{4}$ vi, "while his skin was yellow, mind stupefied, urine scanty, oozing of black, molasses-like blood from mouth and anus, cadaveric odour from body after black vomit; and yet the blood was florid, though with yellow serum, and clot so firm, that, after standing eighteen hours, when four-fifths divided through, the clot could be suspended by one of its sections, without any tendency of the other to break off." Dr. B. adds: "The healthiness of the circulation, simultaneous with a dissolved appearance of the extravasated blood, was not uncommon, and frequently continued during all the successive stages, and up to the hour of death" (p. 84).

of cholera, the plague, and eruptive fevers. It has been pointed out, so far as relates to the fevers of this country, by Drs. Daniel, Drake, Dickson, Stewardson, Stevens, Frick, and others, and has not been neglected by those who have written on the fevers of England, France, Ireland, Scotland, Italy, and Germany, as well as of the West Indies, Algeria, Western Africa, Egypt, Ceylon, and Persia, as the reader may readily find by referring to the pages of Huxham, Cleghorn, Sarcone, R. Armstrong, Evans, Tweedie, Cormack, Stevens, Boyle, J. Davy, Andral, Leonard and Folcy, Clanny, Burns, Stokes, Reid, Stoker, Haspel, Boudin, McWilliams, Jennings, J. Armstrong, Lecanu, Cozzi, Salvagnoli, F. Homes, Clot Bey, C. W. Bell, and Cameron. That it manifests, in the early stage of the disease—in some cases, at least—as it does in other forms of malarial fevers, little or no change from its normal state, may be true; but, in a greater number of instances, it undergoes, even at a very early period—indeed, from the outset, and perhaps, as we shall see, before the attack, and always at an advanced period, alterations of the most important kind, in its chemical composition and physical characters, no one can deny. The fluid is in that state which has received the denomination of hypinosis, and in the worst and malignant cases reaches that denominated spanæmia. The fibrin is frequently less than in healthy blood, or, if it amounts to the normal quantity, its proportion to the blood-corpuscles is less than is found in a state of health. In the early stage of uncomplicated cases, this element is never augmented; sometimes it is in natural quantity, often diminished; but, whatever be the proportion of it at first, the quantity decreases as the disease advances. The blood, from this circumstance, has a tendency towards a state of dissolution, or that which, at a period not very remote from our own, was designated by the name of putrid or adynamic. It presents all the characters resulting from this diminution of fibrin, imperfect separation of the serum and crassamentum, and, as a result of this diminished quantity of the former, a dark-coloured, large, flat, and soft, or over-diffuent coagulum, or no coagulum at all. At the same time, no albuminous coat forms, or, when it does, it is thin and soft, and consists of a gelatinous, parti-coloured deposit on the clot, and the serum is more or less discoloured from the colouring matter of the bile, or from dissolved hæmatoglobulin or blood-corpuscles in suspension. While such are the changes that take place in regard to the fibrin, the quantity of corpuscles is either absolutely increased, or their proportion to the fibrin is larger than in the healthy state; and, in addition, the quantity of solid constituents is also frequently larger than in the normal fluid.

Dr. John Davy states that, in three different examples in which, in the advanced stage of the fever, a minute quantity—a drop or two—of blood was taken from a vein and put into a strong solution of common salt, the corpuscles, under the microscope, exhibited their normal form and about their normal dimensions, excepting in two, in which they seemed thinner than usual. “Whenever the blood was examined, taken from the dead body, however rapidly after death, the corpuscles were seen to be corrugated, as if from incipient putrefaction, to which, after death, and even before, in the advanced

stage, there was a great tendency."¹ Another microscopical inquirer, whose investigations were in like manner conducted in tropical regions, M. de Bienperthuy, observed rather different results. Examining the blood in all stages of the disease, he found that, even at an early period, the parenchyma, which serves as an envelop to the globules, was destroyed. The blood was very fluid, and its colour had changed from a purple to a cherry red. These alterations were greater in proportion to the advanced period of the disease.²

The results obtained in examinations recently made at my request by Professor Leidy, were far from being confirmatory of those last mentioned, and only to a certain extent of those obtained by Dr. Davy. Two portions of blood were examined. The first was procured by me from an individual in the early part of the second stage of the disease, whose skin was of a deep orange colour, and cool; pulse 80, and weak; mind wandering, &c. The second was taken from the heart of the same individual a few hours after death. In both cases the examination was made almost immediately after the fluid was obtained. In the first, the blood was of a dark logwood-red colour, and imparted to the sides of the receiving vessel, as it flowed, a yellowish tinge, resulting from the serum, which was of a deep yellow. There was little disposition to separation or coagulation, and the crassamentum formed was soft and easily broken. Placed under the microscope, the corpuscles were found apparently unchanged. A few only were broken. In a very few minutes, they formed numberless piles, overlapping each, and showing still their flat surface. Soon after, they turned on their edges, and, adhering closely to each other, formed themselves into rouleaux, lying on their side and branching off in all directions. In no case could the slightest indication of the destruction of the enveloping parenchyma, spoken of by M. Bienperthuy, be discovered; so far from this, and so far from being broken up, the corpuscles assumed, in the last particular mentioned, much of the appearance they present in inflammatory blood. Examined fifteen hours after, the corpuscles were found to have lost the disposition to form themselves into rouleaux. They remained separate, and on their flat surface with serrated edges, and soon after, as the process of exosmosis progressed, assumed a granular appearance.

The portion of blood taken from the heart was darker than the preceding, being of a deep maroon colour. It was fluid and homogeneous. Under the microscope, it gave nearly the same results. The globules were much more numerous than in the former specimen. They were entire, and apparently healthy. There was no destruction of the enveloping capsules, and no corrugation, as if from incipient putrefaction. In this case, however, the fluid differed from healthy blood, or from the portion previously examined, in this, that the corpuscles, after appearing detached and showing their flat surface, formed very soon into rouleaux, without passing through the intermediary

¹ Fever of Barbadoes in 1847, '8; Edinb. Journ., lxxii. 281; Ibid, Notes to Blair, p. 85.

² Magne, Ann. Maritimes, vol. iv., for 1844, p. 551.

process of overlapping each other. Other portions were examined a short time after, and at once exhibited the rouleaux appearance, thereby leading to the inference, that these were formed in the fluid before any of it had been placed under the focus of the instrument. The number of these rouleaux was immense, and, branching off at all angles, they formed, as it were, a complete network. On this subject, however, our knowledge has not, so far, made much progress; for though, from its general appearance, and other circumstances, we have reason to conclude that the blood is greatly impaired in the disease under consideration, the microscope has not as yet enabled us to ascertain the exact nature of the changes it has undergone. Few, therefore, who have directed their attention to it will refuse their assent to the remark of an able investigator, that the blood is greatly affected, and its microscopic appearance greatly modified in yellow fever, as well as in congestive and typhous fever; but that it is, nevertheless, not easy to name the specific changes it has undergone.¹

Neither can we say that in regard to the chemical properties of the blood in yellow fever, much satisfactory information has so far been obtained. Dr. Davy informs us that, in several instances observed by him at Barbadoes, during the epidemic of 1847,² the fluid was found acid by the test of litmus paper, thus differing from the blood of other forms of malarial fevers, in which, so far from being acid, it preserves its alkaline reaction. Dr. Davy is not the only one who found the altered blood acid. Trials made by myself prove it to be so, while sometimes it is neutral; and when, bearing in mind the uncoagulated state of the blood, its grumous appearance, and other peculiarities, we revert to the effects of acids upon the fluid—a subject which will be alluded to in another chapter—as well as to the impediment which exists to the excretion of the organic acids which are commonly being formed in the system, and perhaps their introduction through the medium of the efficient cause or poison which, like malarial poisons generally, some chemists regard as acid, we may feel disposed, in the absence of positive facts furnished by repeated and accurate chemical examinations, to admit that the acidity alluded to must often exist in the advanced stage of the disease, and that to it must be ascribed in some measure the changes adverted to.

To Dr. William Stevens, whose work³ appeared in London twenty-three years ago, credit has usually been given for pointing out the fact that the blood, in yellow and other fevers of kindred nature, is deficient in the proportion of its saline ingredients, and that to this circumstance must be referred the dark colour of the fluid observed at some period or another of the disease, sometimes at the onset, always in the last stages. According to his observations and experiments, the red colour of the hæmotosin is imparted to it by the ingredients in question, and any change in the pro-

¹ Microscopic Observations, pertaining to Yellow Fever, by J. L. Riddell, M. D. New Orleans, 1854.

² Edinburgh Journal, lxxii. 280.

³ Observations on the Healthy and Diseased Properties of the Blood. London, 1832.

portion of these will occasion a corresponding change in the colour of the fluid, which remains black even in an atmosphere of pure oxygen, but instantly changes colour when we add to it a clear fluid that contains even a small portion of any neutral salt. There is reason to believe, however, that the facts, on which the theory of Dr. Stevens rests, are not correct, and the theory itself must hence be set aside; and that the saline constituents of the blood, so far from being decreased in amount, sometimes exist in increased proportion. The fact has been well ascertained in respect to cholera, contrary to the assertion of Dr. Stevens, Dr. O'Shaughnessy and others. From this and other circumstances, we may conclude that the diminution of the alkaline reaction in the blood, and its dark colour and diffuent character, are not due to the loss of its salts, but to the impeded elimination of organic acids, which, as already remarked, are constantly being formed in this disease, as they are stated to be in some other zymotic complaints.

But these are not the only changes that are found to occur in the blood of yellow fever. M. Chassaniol, whose residence, during several years, at Guadaloupe, afforded him ample opportunities of investigating the subject, has recently communicated the result of sundry experiments on the blood, instituted at his request by an able chemist, and which led to the discovery that this fluid after death, and most probably in the latter stages of the disease, contains a notable quantity of urea. In one case, 50 grammes of blood, taken from the heart a few hours after death, gave 0.24 of that substance, and, in another, 60 grs. gave 0.29. In stating these results, the operator remarks that the blood examined must have contained a larger quantity of urea than here mentioned, as a portion, doubtless, escaped with the albumen at the moment of coagulation. M. Chassaniol is of opinion that the phenomena of the second stage are due to the poisoning of the blood by this substance.¹ It may not be improper to remark that urea has been detected in the blood of the relapsing fever of Scotland, as also in the serum contained in the ventricles of the brain;² that, in this disease, as in the yellow fever, the urinary secretion is impaired, and that in the former many of the symptoms of the last stage have been satisfactorily traced to the poisoning action of that substance on the blood, and of the latter on the brain and other important organs. Nor should we lose sight of the fact, that urea usually exists in increased quantities in cholera—the amount differing considerably in the several stages of the disease; that it is small in quantity in the intense stage of collapse; increases during reaction, and is in excess when consecutive febrile symptoms occur.³ In this disease, as in yellow and relapsing fever, the urinary secretion is greatly impeded, and we naturally infer that, in all, the presence of urea in the blood is due, in part at least, to want of elimination of it through means of its natural emunctory, and, to some degree, to a disordered condition of the secretory function.

¹ *Compte rendu de l'Ac. des Sci.* Meeting of 12th Dec. 1853.

² Henderson, *Edinburgh Journ.*, lxxii. 223, &c.

³ Reports on Epid. Cholera, Roy. Coll. of Phys., 2d Rep. by D. Gull, pp. 51–3.

In experiments made in this city during the last summer by Prof. Rogers, of the University of Pennsylvania, whose ability and accuracy are well known, the results were somewhat different. Specimens of blood drawn for the purpose of trial during the second stage of the disease furnished no traces of urea. The fluid contained the constituents of bile, and an increased quantity of its usual salts. Neither did other specimens, collected after death, contain urea. Bile again was found, but the salts were in deficient quantity. Assuming that healthy blood presents the following proportions of materials: water 790, and solid matter 210—and that, of the latter, the salts and extractive matter amount to 10.7, the specimen examined contrasted with it to the following extent: the portion taken during life contained water 785, solid matter 215; the salts and extractive matter being 17.5. That collected after death contained water 815, solid matter 185; the salts and extractive being only 8. It is necessary to remark, however, that the negative result, relative to the presence of urea, obtained in the experiments referred to, may not give positive evidence of the absence of that substance, inasmuch as the specimens of blood examined had undergone some slight putrefactive change—as evinced by its peculiar odour—which may have been attended by the destruction of any trace of urea had it been present.

The changes to which attention has been called as occurring in the blood in the yellow fever of both temperate and tropical regions, modified though they may be under different epidemic influences, sometimes in different individuals during the same season, and in the several stages of each particular case, evince the production in that fluid of a morbid condition resulting from the introduction therein of a febrile poisonous agent. They are in no small degree analogous to those produced by the action of other such poisons, as well as of numberless toxical substances of the animal, vegetable, and even mineral kind, and also of putrid substances introduced into the circulation. From this analogy, and other circumstances already adverted to, we not only infer the correctness of the views set forth respecting the cause of the changes in question, but discover reasons for disarding the notion suggested by a fanciful writer, who refers the alteration observed in the blood in the advanced stage of the yellow fever to the absorption of the black vomit.¹ Be this as it may, the fact that the diminution of the fibrin and increase of the corpuscles, as also the diversified proportions of the saline ingredients, and the almost constant dark colour of the blood are observed in other diseases remotely allied to the yellow fever, as well as the absence of any notable or perceptible changes in that fluid in some cases or stages of that fever—so far especially as regards the quantity of the fibrin—clearly proves that we cannot refer solely to that particular condition of the blood all the symptoms of the disease in question. Something else is required for that purpose. All we so far know is, that the specific cause of the fever acts upon the blood in such a way as to destroy its coagulable element. We may apply to the disease before us what M. Andral says of the pyrexia gene-

¹ Audouard, p. 221.

rally. "If the cause acts with little energy, or if the economy resists it, the destruction of the fibrin is not effected. If, on the contrary, the cause continues to act with all its intensity, and the power of the organism fails, the destruction of the fibrin will commence either from the onset of the attack, which is rare, or some time after. This applies as well to typhoid as to eruptive fevers. In all these cases there exists a true intoxication. If it is slight, its effect on the blood doubtless is produced in all cases, but it is not appreciable to our senses; but if the intoxication is more considerable, the effect it produces on the blood becomes perceptible, and is evinced by a diminution in the fibrin."¹

At what period of the disease the changes adverted to commence, it is difficult to say positively. We have seen that, in many cases, they are not perceived in the early stages—the blood retaining its apparently natural aspect—and only become well marked at an advanced period. At the same time, it is difficult to resist the belief that, whatever may be the outward appearance of the fluid—however natural it may seem to us—it is early morbidly acted upon by poison; and thus undergoes from the outset changes which, though escaping our means of investigation, are not on that account the less real. On this subject, I cannot but coincide with a writer who is entitled to our regard in all matters connected with the disease. It is probable, says Dr. Evans, that "the day will arrive when the blood, in all these cases, will be proved to have undergone a particular modification, which our present ignorance prevents us from detecting. It is difficult to understand how, in two cases, apparently similar, and arising from the same cause, one shall exhibit a change in the constitution of the blood, and the other not. Is it not more rational to suppose that in both this change exists, but in different degrees, and that we detect it in the one only because it is evident to our senses?" (p. 247.)

The correctness of the above opinion will appear the more probable when we bear in mind the well known fact that the poison of the fever, floating in the atmosphere during the course of an epidemic, produces an impress on the blood of most, probably of all individuals within the range of its influence, without, however, necessarily affecting them sensibly, or impairing their health. On this subject, the facts recorded by several American and other writers leave no doubt. Alluding to the fever of Baltimore, in 1800, Dr. Potter says: "To ascertain the appearance of the blood in good health, I drew it from five persons who had lived, during the whole season, in the infected parts of the city, who were, in every external appearance and inward feeling, in perfect health. The appearance of the blood could not be distinguished from that of those who laboured under the most inveterate grades of the disease. A young gentleman having returned from the western part of Pennsylvania on the 10th of September, in good health, I drew a few ounces of blood from a vein on that day; it discovered no deviation from that of other healthy persons. He remained in my family till the 26th of the month, and on that day I repeated the bloodletting. The serum had

¹ Essai d'Hématologie Pathologique, p. 68.

assumed a deep yellow hue, and a copious precipitate of red globules had fallen to the bottom of the receiving vessel."¹ Dr. Archer, of Norfolk, has also noticed the same fact, stating as an evidence of the extent to which the predisposing causes of this fever operated, that the blood taken from healthy persons generally exhibited changes which were easily discernible as it trickled down the sides of the basin. These were pretty regularly increased as you approached the infected district.²

In this respect, the poison of the yellow fever approximates to that of other fevers and of zymotic diseases generally. The late Dr. John Mitchell, of Virginia, in the account he left us of the so called yellow fever which prevailed in some parts of that State about the middle of the last century, remarks, that even those who were bled "after a received contagion," before the fever was formed, had a "thin, dissolved, florid blood" even in winter. "This was the constant state of the blood in about thirty or forty whom I have known to have been bled at all seasons of the year."³ "So great and so constant is the difference," says a high authority in reference to the fevers of the Tuscan Maremma—Salvagnoli—"that from the physical examination of the blood only, almost without error the physician may judge if the patient resides constantly in an unhealthy atmosphere, and if the liver and spleen have been altered."⁴ Dr. Stevens makes similar statements relative to the malarial fever of the interior of New York. Dr. S. did not meet with even one intelligent practitioner in the Genesee country who was not aware of the fact that the blood of the inhabitants, during the sickly months, is very different from that of individuals who arrive from healthy situations: "Whilst even in those who reside in the most unhealthy situations, and who have not yet had the fever, it is not only dark in colour, but evidently so much diseased in its properties as to be very unlike the blood in health."⁵ The same writer has noted the changes during the prevalence of the malarial fevers in the West Indies.⁶ In an interesting report on the epidemic ague, or fainting fever of Persia, which occurred in Teheran in the autumn of 1842, Dr. Ch. W. Ball informs us that, while the disease was at its height in the town, the blood even of those not sensibly attacked, was universally of a dark, dusky, reddish-brown colour, very different from that of healthy venous blood; and that in general the serum did not separate from the clot.⁷

It may be true that those cases in which the blood remains apparently unaltered, and especially those in which, instead of being dark coloured at the outset, it is of a bright florid hue, militate against the views here set forth respecting a commencement of deterioration of that fluid anterior to the attack. But, as was said before, there are reasons to think that, though natural and healthy in appearance, it must, nevertheless, have undergone morbid

¹ A Memoir on Contagion, p. 54.

² Hist. of the Yel. Fev. of Norfolk in 1821, Med. Rec., v. 68.

³ Med. and Philos. Register, iv. 188.

⁴ Saggio illustrativo le Tevole della Statistica Medica della Maremma Toscana, p. 211.

⁵ On the Blood, pp. 216-219.

⁶ Ibid., pp. 214-217.

⁷ British and Foreign Med. Rev., xvi. 521.

changes, which, if the downward course of the disease be not arrested, will go on increasing till they have attained the degree that marks the latter stage; whilst, in regard to those instances characterized by a florid red appearance of the blood, the effect may be ascribed to the strong efforts made by the powers of life to resist the deadly action of the poison and to eliminate it from the system; in other words, to the excess of reaction which follows the first impression of the poison, in virtue of which a flame is kindled, destined, if the powers of life succumb, to die out in a few short moments, leaving the system at the mercy of the deadly foe. Certain it is, that the peculiar appearances in question are found in the inflammatory form of the disease, when symptoms of reaction are predominant and the circulation is greatly accelerated; and that, as these symptoms fade away, this florid redness diminishes, and gives place to the dark colour and diffuence of the blood; while in the true, malignant, or congestive variety, when the reaction is feeble, or does not exist, we may expect the blood to be at the very outset dark coloured, emitting the peculiar smell noticed, evincing a deficient power of coagulation, presenting a soft clot, rarely, if ever, covered with a crust, and from which a large quantity of black colouring matter falls to the bottom of the cup, with deep-yellow or orange, but never bright-red, coloured serum, and exhibiting in the latter stages, when the disease proves fatal, and sometimes very early in bad cases, the thinness, blackness, already noticed, as well as a deficiency in amount of animal matter and sometimes of saline ingredients. To this let me add that, in the yellow fever, the prompt and firm coagulum, and especially the buffy coat and the cup-like form of the latter, will probably be found, as it is in other pyrexia or zymotic diseases generally, only in cases complicated with a local inflammation, a morbid state, which may be, and often is, appended to, but forms no necessary part of, the disease; in pure cases of which the fibrin, though generally diminished in quantity, may sometimes, and at a certain period of the case, be in natural proportion, but is never in excess, as in the phlegmasia.

As to the intimate nature of those changes we know but little. We do know, however, that the lessened power of coagulability, and the dark coloration of the blood, are combined with, or are ascribable to, a deficiency of animal matter, or a derangement in its elementary principles, as well as to an accumulation of organic acids; and that to an increase of saline matter may be referred the floridness of the blood in the early stage of some forms of the disease. We know, also, that the yellow or red colour of the serum is owing partly to the colouring matter of the bile, and partly to dissolved hæmatoglobulin; and that the peculiar smell is probably produced by the evolution of a volatile salt of ammonia, as conjectured by very competent authorities, is also very true. But, how the poison of the yellow fever produces those effects, and whether, when it has done so, it remains in the circulation until eliminated through some of the excretories, or enters into a peculiar chemical combination with the blood, are points which must be deferred for future inquiry. All that need be said in this place is, that, as in other fevers of a

kindred, though not identical character—the remittent of the African coast,¹ and malarial fevers generally—as, also, in typhus, typhoid, and relapsing fevers, in cholera asphyxia, in eruptive diseases, in the morbid states induced by certain vegetable and animal poisons, the blood presents phenomena not very dissimilar to those noticed in the yellow fever; and as, nevertheless, each of the diseases mentioned presents symptoms which impart to it a character of individuality that enables us to distinguish it from the others, we may infer that those changes are of a secondary nature; or that the poison, which produces the effects in question, floats in the blood, and occasions, through its means, those modifications on the tissues which constitute the disease under consideration; for it is impossible to suppose that the blood, though presenting appearances externally analogous in those diseases, can be really similarly constituted, and yet occasion morbid states so specifically different.

CHAPTER VI.

CIRCULATORY SYSTEM CONTINUED—ACTION OF THE HEART AND ARTERIES.

PULSE.—From the description I have presented of the symptoms of the yellow fever of Philadelphia, it will be seen that considerable differences exist, in different cases, in regard to the action of the heart and arteries, as manifested in the pulse. These differences are observable during the several periods or stages of the disease, though more particularly during the first. They show themselves in the various forms or varieties of the disease, which, indeed, they mainly serve to characterize, each form being generally accompanied by a peculiar condition of pulse; and as those forms vary, in point of frequency, in different epidemic seasons, in various situations and in the same place, and, during the same epidemic, at different phases of the latter and in different subjects, so the modifications in question necessarily vary in point of frequency with the accompanying circumstances. They have reference to the degree of strength and fulness of the pulse, to the quickness and rapidity of the pulsations, and to the peculiar character of the rhythm.

a. Strength.—We have seen that, at the commencement of the attack, when the period of reaction has fairly set in, the pulse is often strong, tense, and full, and even bounding. This was especially the case during the memorable epidemic of 1793, as we find recorded in the writings of Rush (iii. 53), Currie (p. 20), Catlirall (pp. 23, 24), Deveze (p. 23), Barnwell (p. 372), Nassy (p. 19). The same peculiarity was observed, to a greater or less extent, in the character of the pulse, during the subsequent epidemics of this city in 1794, 1797, 1798, 1802, 1805, 1820,² and 1853.

¹ Bryson, Ed. J., lxix. 136; McWilliams, p. 148.

² Rush, iii. 203, iv. 10, 40; Currie, Bil. Fev., p. 219; Firth, p. 26; Caldwell, Fev. of 1805, p. 81; S. Jackson, pp. 59–62.

The peculiarities in question of the pulse are characteristic of the inflammatory variety or form of the disease, and exhibit themselves in bolder relief in proportion to the energy of the reaction and the grade of violence of that variety. It has been noticed in all other cities or towns of the United States. Among the characteristics of the fever of New Orleans, Thomas (p. 83) mentions "un pouls généralement très dur et développé." Girardin (p. 33) speaks of a full, strong, tense, and regular pulse; Gros (p. 9), of a full, strong, and regular pulse; and Barton (p. 16), Harris (*Am. Journ. of Med. Sci.*, xiv. 41), Baxter (*Reposit.*, xxi. 3-12), *Report of Fever of 1839* (p. 331), and Harrison (*N. O. Journ.*, ii. 132), furnish us with similar statements; while many others bear testimony to the same effect in regard to the fevers of Norfolk, Wilmington (N. C.), Key West, Natchez, Galveston, Charleston, Baltimore, New York, New London, Boston, and New Haven.¹

Nor do we find this character of pulse unknown in the yellow fever of Europe. In the fever of Cadiz, in 1800, the pulse, during the period of reaction, was often strong, full, and hard; and the subsequent epidemics of that and other cities of Spain, as well as of Gibraltar, exhibited the same phenomena.² Boyd found it of that kind in the fever of Minorca;³ and, in Leghorn, in 1804, the strong, hard pulse was generally encountered—"i polsi duri, stirati, tesianche nelle remissione della febbre"—says Tommasini.⁴ Dufour, as remarks the same celebrated writer, found the pulse generally tense and full; Palloni, hard and frequent; the medical commissioners sent from Lucca to Leghorn on that occasion, say it was frequent and vibrating; Pasquetti and Brignole say that, during this stage (the first), the fever is ardent, and that the pulse begins to lose strength only at the accession of the second stage. La Coste was the only one who found the pulse weak and depressed in the first stage.⁵

Dr. Rochoux regards the strong, full, large, and developed pulse as characteristic of the yellow fever of the West Indies;⁶ and the medical inquirer

¹ Addoms, p. 9; Valentin, p. 165; Archer, *Record.*, v. 66; Hill, *ib.*, v. 89, 90; Ticknor, *N. A. Med. and Surg. Journ.*, iii. 223; Merrill, *Med. and Phys. Journ.*, ix. 244; Cartwright, *Recorder*, ix. 13; Hogg, *Western Journ.*, i. 416; A. Smith, *Am. Journ. Med. Sci.*, xxv. 501; Lining, *Edinb. Essays*, ii. 411; Moultrie, p. 3; Irvine, p. 28; Shecut, p. 119; Dickson, *Med. and Phys. Journ.*, iii. 254; Drysdale, *Med. Mus.*, i. 29, 122; Revere, *Recorder*, iii. 224; Kelly, *Am. Journ. Med. Sci.*, N. S., xiv. 377; Seaman, *Webster's Col.*, p. 7; E. H. Smith, *ib.*, p. 116; Munson, *ib.*, p. 180; A. Hosack, pp. 11, 12; Stone, *N. O. Journ.*, v. 560; Warren, in Tytler, p. 501; Lathrop, *Med. Repos.*, ii. 473; De Rosset, *ib.*, ii. 153; Wragg, *Charleston Journ.*, x. 74; Lewis, *N. O. Journ.*, i. 297.

² Caisergues, p. 168; Doughty, p. 113; Berthe, p. 85; O'Halloran, p. 79; Fellowes, pp. 52, 200; *Edinb. Journ.*, i. 448; Smith, *ib.*, xxxv. 42; Amiel, Johnson, p. 263; Louis, pp. 208, 209, 211, 212; Pariset, pp. 29, 375; Laso, *Periodico de la Sociedad Médico-Chirurgia*, vol. ii. No. 3, p. 343, quoted by Rochoux, p. 491.

³ See Johnson on *Trop. Cl.*, p. 299.

⁴ *Febbre Gialla*, i. 154.

⁵ *Ibid.*, § 9, p. 11. See also Palloni, p. 4; *Med. Repos.*, viii. 426; Dufour, *Marseilles Journ.*, iv. 51.

⁶ *Rech. sur les Diff. Mal.*, &c., p. 313; *Ib.*, *Rech. sur la Fièvre Jaune*, p. 135.

will find, on reference to most writers on the diseases of tropical climates, that this physician's statement will apply to a large, if not to the largest, number of cases; while, in others, it is full, more or less throbbing, but soft.¹ In some instances, the pulse is small and hard. Moultrie noticed this peculiarity a century and more ago (p. 3), and it has attracted the attention of many since his time.

From the weight of authorities referred to in reference to the occurrence of a strong, hard, and full pulse in the yellow fever of this country and other localities of temperate regions, I might reasonably conclude, even could I not appeal to personal experience, that the reality of such a condition of the arterial circulation is placed beyond the possibility of doubt. By some writers, however, the fact has been viewed in a different light, and they have not hesitated to call in question the correctness of the statements made on the subject. Dr. Tully, of Middletown, is disposed to do so, affirming that the same pulse is often described, by different physicians, not only under a variety, but under opposite denominations, and that he has often witnessed a gaseous mistaken for a strong and full pulse; a wiry pulse, indicative only of irritation, for a hard one; and a *morbidly* natural pulse for the genuine pulse of health. And Rochoux, in the same spirit, but with a different object in view, indulges in kindred remarks: "Sometimes I have found it a little stiff and tense; but even then it was easily compressed. It is doubtless to cases of this kind, observed without sufficient attention, that we must attribute what certain authors have said of the strength of the pulse in the amaril typhus." Such denials are easy, and are convenient for those who endeavour to establish a favourite hypothesis. But it will be difficult for the above American and French writers, whose experience was circumscribed, in the one instance, to a dozen cases, and in the other, to a larger number, but to a single epidemic, and who, besides, had a theory to support, to persuade the unbiassed and well-informed reader that all preceding observers have been deceived on the subject of the pulse, and that they alone are entitled to belief.

But although the first stage of the disease is sometimes, and, in some seasons and places, often, characterized by a condition of pulse such as has been described, it frequently happens that the attack commences in a different

¹ Pugnet, p. 354; Fontana, p. 72; Lefort, De la Saignée, &c., p. 577; Jackson, Fevers of Jamaica, p. 280; Ib., Sketch, p. 46; Blanc on Seamen, p. 413; Gillespie, p. 38; Barrington, Am. Journ. Med. Sci., xii. 311; Bancroft, p. 30; Moseley, p. 436; Osgood, p. 9; Desportes, i. 193; Imray, liii. 79; McArthur, in Johnson, p. 346; Poissonnières, p. 64; Ralph, Ed. Med.-Chir. Tr., ii. 70; Wallace, Edinb. Journ., xlv. 275; Savarésy, p. 270; Peixotto, N. Y. Med. and Phys. Journ., i. 412; Gilbert, p. 65; Dariste, p. 158; Wilson, pp. 8, 9; Bally, p. 220; Dancer, Med. Ass., p. 82; Maher, p. 848; Caillot, p. 17; Hunter, p. 64; Davidson, Med. Repos., viii. 248; Morgan, Med. and Phys. Journ., iv. 4; Pym, p. 228; Dyott, Med.-Chir. Rev. and Journ., iv. 1003; Belcher, Edinb. Journ., xxiii. 249; Warren, p. 9; Lefoulon, p. 68; Bruce, 278; Levacher, p. 72; Hillary, pp. 147, 150; Madrid, pt. ii. p. 24; Rufz, p. 13; Gillkrest, p. 271; Leblond, p. 103; Rouppe, p. 304; Ib., Transl., p. 408; Cayenne in 1850, p. 176; Lallemand, Fev. of Rio Janeiro, p. 79.

and opposite way—the pulse being soft or feeble, though more or less full; or weak, depressed, and small; and, at times, natural. These conditions, particularly the last, indicate imperfect or deficient reaction, and are generally characteristic of the congestive or malignant variety of the disease. Such peculiarities of pulse are observed, to a greater or less extent, in all epidemics, and are found to preponderate, or to be almost universal, in certain seasons and places. They were noticed here in 1793, when, as Dr. Rush remarks, the fever came on with a weak pulse, often without any preternatural frequency or quickness, and sometimes so low as not to be perceived without pressing the artery at the wrist. (Vol. iii. p. 53.) Dr. Rush also speaks of a full, soft pulse, imparting the impression of the artery being filled with air.¹

The same characters of pulse—the weak and full, the depressed and small, and even the gaseous—were noticed in our subsequent epidemics, and especially in 1794 and 1799. Dr. Caldwell states that, in 1805, cases occurred in which there was no reaction or febrile commotion; the pulse and other functions or organs remaining undisturbed (p. 86). They were likewise noticed in 1820, and constituted a striking feature in many, if not the larger number, of the cases which occurred in 1853 and 1854. It may not be improper to state here that, in instances in which the pulse disappeared readily under the pressure of the finger, and other symptoms denoted a depressed condition of the system, the heart was found to beat with energy and convulsively. (*Caldwell*, pp. 85, 86; *Jackson*, pp. 52–57.)

The characters of pulse above described have been often referred to in the descriptions of the epidemics of New York, Baltimore, Middletown, New Haven, Natchez, Charleston, Wilmington (N. C.), Mobile, New Orleans, and Norfolk.² In a total number of observations amounting to 1771, in the three stages of the disease, made at the Roper Hospital, Charleston, in the sickly season of 1854, it was found that the pulse was full in 193, small in 263, weak in 340, and strong in 79. “An inspection of the column will show the important fact,” remarks the reporter, “that want of energy and vigour were the most frequent characteristics of the circulation.”³

Roehoux, who, under the name of amaril typhus, has described the only yellow fever of temperate climates he ever had occasion to observe, that of Barcelona, and holds it up as the legitimate type of the disease called yellow fever in this country and Europe, but which differs in toto from the fever of hot latitudes, affirms, that the pulse in it is always weak, unequal, and irregular; and though we have reason to believe he was as exclusive in this as on other points, there is little doubt that, on the occasions mentioned, such, in most cases, was the character of the pulse. Dr. R. Jackson, a much better

¹ See Rush, iii. 53; *Ib.*, iv. 10–40; Deveze, pp. 28, 29; Barnwell, p. 372.

² Drysdale, i. 29; Revere, iii. 224; E. H. Smith, p. 116; C. Drake, xxi. 134; Townsend, p. 146; Tully and Miner, p. 297; Perlee, iii. 11, 12; Munson, p. 180; Irvine, p. 30; Dickson, iii. 254; Hill, v. 90; Archer, v. 61; Gros, p. 9; Baxter, xxi. 3; Barton, p. 9; Kelly, xiv. 379; Lewis, N. O. Journ., i. 296; Hayne, Charleston Journ., vii. 5, 6; Copland, iii. 141; Lallemant, p. 79.

³ Wragg, Charleston Med. Journ., x. 74.

authority on all matters of the kind, remarks, that in Spain, in 1820, the pulse was often small, frequent, and irregular during the period of invasion, sometimes sluggish and obscure, rarely, if ever, buoyant and elastic (p. 71). In 1800, also, it was sometimes of a kind denoting deficiency of energy in the action¹ of the heart. So, likewise, was the condition of the pulse at Malaga in 1804,² and at Gibraltar at different periods.³

Nor is it alone in temperate climates that the peculiarities of pulse in question, and the form of the disease they serve to characterize, are observed. That, by some writers, an assertion to the contrary has been ventured; that they maintain that, in the yellow fever of tropical regions, the first stage is always marked by a condition of pulse indicative of high inflammatory reaction, is true. But true it is also, that such a statement, made to uphold a favourite hypothesis, will, on examination, be found as void of foundation as that of the invariable absence of the strong, full, and hard pulse in the fever of temperate regions. The fact, recorded already, of the frequent occurrence in hot climates of a typhoid congestive form of the disease characterized by imperfect, broken, or defective reaction, would alone suffice to show the erroneousness of the statement in question, since the existence of such a typhoid and congestive condition of the system is incompatible with that of a strong, full, and throbbing pulse. But the fact of the existence of the contrary condition of pulse in the fever of tropical climates, and the additional proof therefrom derived, of the identity of that disease with the yellow fever of this country and Europe, may be found in the positive statements of writers on the former. Hillary (p. 147) tells us that, in some cases, the pulse in the stage of reaction is soft, low, and oppressed. Bancroft (p. 30) says it is sometimes oppressed and irregular. Moseley (p. 436) states that, in some cases, it is low, vacillating—sometimes deeply depressed. Imray (lii. 79) mentions that, in general, it is weak, sometimes scarcely above the natural standard. Lempriere (ii. 85–6) states that the pulse, in his hybrid variety, has an irregular, creeping motion, and none of the symptoms which seem to denote an inflammatory affection. Frost (xiii. 29) remarks that, in some patients, the pulse was unnaturally depressed and weak. Wilson (pp. 11–19) states that, in the congestive variety, the pulse varied much in frequency and expansion, but was always weak and yielding. In some forms of the disease, described by Dr. R. Jackson (p. 91), the pulse is represented as being “seldom much changed from the pulse of health, unless by marked deficiency of quickness and energy in the mode of pulsation.” Madrid (p. 24), Wallace (xlvi. 275), Makittrick (p. 91), Vatable (p. 345), Bruce (p. 278), Pym (p. 228), Dyott (p. 1003), and Furlong (p. 290), state that the pulse, though full, is soft and easily compressed; while Comrie (*Ed. Journ.*, xiii. 167–175), Caillot (p. 19), Maher (pp. 842–867), Pym (p. 233), Davidson (*Repos.*, viii. 249), Dubreuil (*Journ. Univ.*, viii. 333), Arnold (p. 8), J. Clark (p. 10), Rufz (p. 11), Moseley (p. 439), and Catel (p. 11),

¹ Caisergues, p. 168; Berthe, p. 84.

² Fellowes, p. 200; see *Ed. Journ.*, i. 448.

³ T. Smith, *Ed. Journ.*, xxxv. p. 42; Gillkrest, *Cycl. of Pract. Med.*, ii. 272.

furnish additional evidence of the facts contended for. Barry tells us that, at Sierra Leone, in 1823, the pulse was hurried and somewhat full, but seldom indicated any degree of inflammatory action.¹

Indeed, cases, unattended with arterial excitement, were noticed from the earliest times; for Father Labat tells us (vol. i. p. 73) that the opening symptoms of the disease were sometimes succeeded by a high fever (*grosse fièvre*); at others, by an internal fever, which did not manifest itself outwardly. In such cases, the pulse could not very well have been full, bounding, and strong. The contrary condition of the pulse, in like manner, attracted the notice of Warren, who, after stating that it is, in some, quick, high, and throbbing, adds: "In others, it is quick, low, and vacillating" (p. 9).

b. Frequency.—Many of the writers who have treated of the yellow fever of this city and country, as also those who have described the fever as it occurs elsewhere, have spoken more or less pointedly of the frequency of the pulse in the majority of cases, contenting themselves, however, with the simple enumeration of the fact, without specifying the average number of pulsations. From the adoption of this course, we may infer that the frequency to which they allude did not transcend the usual rate observed in febrile complaints generally. We have seen, in the foregoing description of the disease, that, in the inflammatory variety, whether of the violent or milder grade, the pulse has been stated to vary from 80 to 120 or more in a minute; being, as Dr. Rush remarks, as frequent as in pleurisy and rheumatism. Barnwell (p. 369), states it to have ranged, in 1793, from 120 to 130. In 1820, it varied in such cases from 100 to 120. Similar observations were made in 1853 and 1854. In New York, in 1822, it ran from 90 to 120. During the epidemic of the same city in 1795, Dr. Seaman states it to have been from 80 to 130.² Louis (p. 208) never found it to exceed 100 at Gibraltar in 1828, whatever might be the issue of the case. In the West Indies, the frequency is much the same. Dariste tells us that, at Martinique, the pulse varied from 100 to 110. Savarésy (pp. 270–9) from 80 to 90. Ralph, in one variety, found it seldom above 100. In another, it rose to 120 (p. 68). Dr. Steward, in his account of the fever of Grenada, states that the pulsations did not exceed 100 in a minute.³ Rufz states them to have ranged between 100 and 112.

In a few cases of a decidedly inflammatory kind; in many in which this character is questionable; and still more frequently in those of a typhoid or congestive form, the pulse has been either slower or more rapid. In 1839, at New Orleans, it was generally, in cases of the latter kind, at 120.⁴ In his letter to Mr. Vance, Dr. Pym states that the pulse rose at the commencement as high as 140 (p. 59). In the fever of 1804, it often reached to 130, while, in his letter to Sir R. Keith, he mentions the range to have been only from 100 to 120. In this country, it has been found occasionally to be preternaturally

¹ Report in Boyle on Diseases of Western Coast of Africa, p. 271.

² Med. Rep., iv. p. 249.

³ Med. and Phil. Register, iii. p. 184.

⁴ Report, p. 331.

quick and frequent—particularly in cases where it was weak. Dr. Rush (iii. p. 203) saw it as high as 140, or even 176, in a minute. This occurred in 1794. In 1820 it rose, in one instance, to 160 strokes in a minute. (*Jackson*, p. 59.) According to Dr. Rochoux, who had in view the fever of Barcelona in 1821, the pulse at the commencement is characterized by great velocity, reaching usually to between 120 and 140 in the minute (p. 490), and never falling below 100. In tropical climates, also, we hear of its presenting the same character. In Comrie's account of the ardent fever of the West Indies, the pulse is represented as being sometimes at 130,¹ and weak; sometimes between 130 and 150, and even higher—small and weak. Chisholm has known the pulse to rise as high as 130—but never higher—nor slower than 30 (i. 162). At Cayenne, in 1850, the pulsations reached as high as from 120 to 160 (*Report*, p. 176), and Finlay notes them, as a general occurrence, at 130 (p. 12), whilst Lallemand has seen them reach 160 and even 180 (p. 78). Other writers, in temperate and tropical regions, have called attention to the quickness and frequency of the pulse.²

Such instances, however, are not always encountered. In many cases, the tendency is rather towards an uncommon slowness. Deveze, in some cases, found it very slow (p. 28), and Dr. Rush, in 1793, notices such instances as of not unfrequent occurrence (iii. 54). In 1794, it was seldom very frequent. In one case, it beat 64 strokes in a minute for several days, and at no time exceeded 96. In the majority of the cases which came under Dr. Rush's notice that year, where the danger was great, the pulse seldom exceeded 80 strokes in a minute (iii. 203). Dr. Caldwell tells us that, in 1805, he found it natural in some instances (p. 86).

Dr. Townsend states that in New York, during the epidemic of 1822, the pulse was "sometimes as low as 80, and even 60 and 55 in a minute from the beginning of the attack." In one case, it was as low as 45, six hours after the invasion of the disease, and never became more frequent than 70 (p. 146). Drysdale found it as low as 50 in Baltimore (i. 123). Barton as low as 40. Dr. J. Clark remarks (p. 7) that, in Dominica (1793), the pulse never rose higher than 90. In Sierra Leone, the pulse, in many cases, was considerably below par, presenting a peculiar sluggish action known to be demonstrative of debility (*Boyle*, p. 290).

In fact, the slow, sluggish, or natural pulse has been frequently noticed

¹ *Edinburgh Journ.*, xiii. 167, 170, 175.

² *Pariset, Observ.*, p. 29; *A. Smith, Am. Journ.*, xxv. 212; *Amiel, Edin. Journ.*, xxv. 263; *A. Hosack*, pp. 11, 12; *Palloni*, p. 4; *Tommasini*, i. 11; *Caldwell*, p. 81; *Doughty*, p. 113; *Smith, Edinb. Journ.*, xxxv. 42; *Moultrie*, p. 6; *Hill*, v. 89, 90; *Barton*, pp. 9, 10; *Shecut*, p. 119; *Harrison*, p. 132; *Stone*, v. 560; *Wragg, Charleston Journ.*, x. 74; *Gilbert*, p. 65; *Dancer*, p. 82; *Dyott*, p. 1003; *Morgan, Med. and Phys. Journ.*, iv. 4; *Maher*, pp. 842, 848; *Pym*, p. 228; *Fontana*, p. 72; *Pugnet*, p. 354; *Vatable*, p. 345; *Rufz*, p. 11; *Wilson*, pp. 8, 13; *Gillespie*, p. 38; *Barrington*, xii. 311; *Bancroft*, p. 30; *Peixotto, New York Med. and Phys.*, i. 412; *Osgood*, p. 13; *Levacher*, p. 72; *Hillary*, p. 147; *Moseley*, p. 436; *Imray*, liii. 79; *Hunter*, p. 64; *Savarésy*, p. 270; *Warren*, p. 9; *Bruce*, p. 278; *Ralph*, ii. 65, 70, 72; *Makittrick*, p. 95.

during the first stage in the yellow fever of temperate and tropical regions, and is mentioned by almost every writer of note on that disease.¹ So general, indeed, is this tendency in the less inflammatory and in the congestive forms of the fever, and so unusual is the great rapidity of the pulse in these or other forms, that we might, from that circumstance alone, infer that those who regard extreme rapidity as the usual characteristic of the pulse in the fever of cool regions, have erroneously converted the exception into the general rule. But, on this point, as on many others, we may controvert the assertion by an appeal to authorities. Bally, François, and Pariset, who observed the epidemic described by Dr. Rochoux, state that the pulse ranged from 80 to 90, and never rose to 100 (p. 394). The same may be said of other epidemics of Europe; Berthe (pp. 84, 85), Caisergues (p. 168), Palloni (p. 4), O'Halloran, (pp. 77-126), Jackson (p. 81), and Blin (p. 6) speak, it is true, of an accelerated, but say nothing of an excessively rapid pulse; and still less do they deny the occurrence of a natural or slow pulse. Lonis, as we have seen, did not find it ever to exceed 100 strokes at Gibraltar in 1828, and Gillkrest (p. 271), who saw the disease on that occasion, represents a rapid pulse as a possible contingency, but not as a necessary occurrence.

Such, as I have described, are the usual varying characteristics of the pulse during the first stage of yellow fever, as regards activity, fulness, strength, and frequency. It may be proper to add, that while these several differences are, at times, found to mark the disease during the whole of some epidemics, on other occasions they appear intermixed in almost equal proportions. In such cases, it is not unusual to find the pulse varying considerably at the different phases or periods of the epidemic. The change was noticed in this city in 1793. In August, and during the first ten days of September, a small, irregular, and slow pulse prevailed very frequently. After that period, it was less commonly noticed, and, in proportion as the weather became cool, it gradually disappeared, and was followed by a full, tense, quick pulse. (*Rush*, iii. 54-56.) Drysdale made a similar observation, in Baltimore, the year following (i. 123).

Nor is it less true that such differences are found to occur not only at different periods during the course of the same season, but at the same time in the different parts of an infected place. Indeed, the circumstance might be inferred from the fact already mentioned, that the several forms of the disease—which are, in great measure, characterized by different conditions of pulse—are often variously preponderant in the several localities of such places.

In not a few instances during the several epidemics by which the city of Philadelphia has been visited, the pulse exhibited great irregularity in respect to its rhythm. Speaking of 1793, Dr. Rush states that the pulsations often intermitted after the fourth, in some after the fifth, and in others after the

¹ Dalmas, p. 6; Gillkrest, ii. 271; Doughty, pp. 122, 128; Comrie, p. 175; Hillary, p. 147; Frost, xiii. 29; Ralph, ii. 75; Lefort, op. cit., p. 569; Lallemant, p. 79; Imray, liii. 79; Moseley, p. 436; Munson, Webster's Collect., p. 180; Seaman, ib., p. 7; O'Halloran, p. 126; Arnold, pp. 124, 128; Dickson, iii. 254; Irvine, p. 23; Harrison, p. 133; Waring, p. 46.

fourteenth stroke. "These intermissions occurred in several persons who were infected, but who were not confined by the fever. They likewise continued in several of my patients for many days after their recoveries" (iii. 53). This disposition to intermission was combined with a depressed and slow condition of the pulse. It was common in 1794, in 1797, and has been observed at all subsequent sickly periods (iii. 203; iv. 10). Drysdale (i. 122) found it hobbling—a full pulsation being succeeded by another of less force, and no two successive strokes resembling each other in frequency. In one person, he counted three pulsations in one-sixth of a minute—in another sixth, they amounted to sixteen. In many cases, there was an intermission after the 2d, 3d, 4th, 5th, 6th, or 14th strokes. It not unfrequently happens that two rapid pulsations are followed by an intermission of greater or less duration. This again is succeeded by two pulsations similar to the others, and so on. This condition of pulse is often connected with powerful contractions of the cœliac artery. The intermittent or irregular pulse has been noticed by many others;¹ but Chisholm mentions, as a remarkable circumstance, that the pulse, at Grenada, never intermitted even at the approach of death (i. 162).

From the details into which I have entered, the medical reader will perceive that there is little or nothing in the condition of the pulse which may be viewed as characteristic of the yellow fever, and nothing that is peculiar to that disease, unless, perhaps, it be the tendency it very often exhibits, amid a train of alarming symptoms, either to retain its normal condition in point of volume, strength, and frequency, or to be depressed in every respect below the standard of health. Even in cases in which inflammatory reaction takes place, it differs little, in point of energy, from that of other febrile diseases of like character; or, if it does, the difference lies in its being less frequently tense and hard; while rising, as it seldom does, to 120—being generally under 110, and often under 100—it cannot be viewed as distinguishable for its rapidity. It is doubtful, moreover, whether the tendency alluded to above—to retain the natural standard, or to be reduced in the early stage below that standard—can be adduced as a distinctive peculiarity of the yellow fever, or as one of its main pathognomonic characters, inasmuch as it is not manifested in all cases, and is found to occur in other diseases, but remotely connected with the former.

But if nothing distinctively peculiar can be noted in the pulse during the first stage or period of the yellow fever, matters assume, in the large majority of cases, a different turn at the accession of the metaptosis or remission, which, as we have seen, takes place after the disease has progressed a short time. For then, whatever may have been the degree or kind of deviation which the pulse exhibited—however strong, hard, accelerated, or quick it

¹ Caillot, p. 19; Maher, p. 867; Davidson, *Med. Repos.*, i. 166; Madrid, pt. 2, p. 24; O'Halloran, p. 126; Savarésy, p. 277; T. Smith (*Edinb. J.*, xxxv. 42; Waring, p. 9; Ralph, ii. 65, 68; Levacher, p. 74; Pugnet, p. 354; Lemprière, ii. 62, 83, 85; Seaman, p. 7; Hayne, *Charleston J.*, vii. 5, 6; Lallemand, *Fever of Rio Janeiro*, p. 79.

may have been during the period of febrile commotion—and whatever may be the issue of the case, it almost suddenly loses all these morbid characters, assumes once more the normal standard, and, in not a few cases, even becomes slower than in health; sinking from its former rapidity to 90, 80, or 70; in other instances to 60, 50; and, in a few, to 40, or even 30 strokes in a minute. In the various epidemics of this city, this reduction of the pulse at the period in question, and even the more striking instances of it alluded to, have been observed;¹ and if we open the description of the disease as it has prevailed and continues to prevail in other parts of this country, in Europe, and in tropical regions, we shall find that it constitutes one of the most important features of the disease. Such has been the case in Baltimore,² Norfolk,³ Alexandria,⁴ Charleston,⁵ Savannah,⁶ New Orleans,⁷ Natchez,⁸ Galveston,⁹ Mobile,¹⁰ New York,¹¹ Middletown,¹² Boston.¹³ Such, also, has been the case in Cadiz, Barcelona, Seville, and other cities of Spain,¹⁴ in Leghorn,¹⁵ and Gibraltar;¹⁶ and there is scarcely a writer on the fever of hot climates who has not dwelt more or less pointedly on the change.¹⁷

In fact, so constant and marked is the latter everywhere, that it may safely be viewed as a characteristic feature of the disease, and as the same phenomenon is observed in no other form of fever—except, perhaps, as an accidental occurrence—it may be regarded as pathognomonic and distinctive. As to the period at which it occurs, some differences are found in different

¹ Rush, iii. 54, iv. 13; Currie, p. 26; Cathrall, pp. 30, 26; Barnwell, p. 373; Deveze, p. 23; Firth, p. 26; Jackson, pp. 56, 7, 60; Caldwell, p. 84.

² Revere, iii. 225.

³ Valentin, p. 167; Archer, v. 67.

⁴ Dick, Med. Reposit., vii. 192.

⁵ Lining, ii. 417; Moultrie, pp. 9, 12; Dickson, iii. 255; Irvine, pp. 29, 30; Hayne, Charleston J., vii. 56.

⁶ Waring, p. 45.

⁷ Thomas, p. 83; Gros, pp. 10, 12; Girardin, p. 35; Fever of 1820, p. 9; Ib. of 1839, p. 333; Harrison, p. 132.

⁸ Cartwright, ix. 10.

⁹ Ashbel Smith, Am. J., xxv. 501.

¹⁰ Kelly, Am. J., N. S., xiv. 377, 8.

¹¹ Seaman, Med. Reposit., iv. 249; Townsend, p. 148.

¹² Tully, pp. 297, 8.

¹³ Rand., Med. Repost., i. 473.

¹⁴ Pariset, Fev. of Barcelona, pp. 400, 427; Ib., Fev. of Cadiz in 1819, pp. 10, 30; Rob. Jackson, pp. 74, 92; O'Halloran, p. 83; Audouard, p. 59; Berthe, p. 88; Caisergues, p. 168; Rochoux, p. 491; Jourdain, An. de la Méd. Phys., v. 257; Edinb. J., l. 448.

¹⁵ Palloni, p. 5; Dufour, Marseilles J., iv. 51.

¹⁶ Amiel, in Johnson, p. 264; Gillkrest, ii. 270; Louis, p. 211.

¹⁷ Warren, pp. 10, 15; Levacher, p. 74; Wilson, pp. 10–14; Peixotto, N. Y. Med. and Phys. J., i. 413; Pugnet, p. 354; Dubreuil, Journ. Univ., viii. 333; Bruce, p. 278; Fontana, p. 73; Vatable, p. 345; Jackson, Tr., p. 260; Ib., Sketch, i. 70, 110; Blane on Seamen, pp. 413, 415; Gillespie, pp. 40, 48; Hillary, pp. 149, 151; Brancroft, p. 32; Desportes, i. 194; Lempriere, ii. 61, 2; Imray, liii. 79, 80; Osgood, p. 11; McArthur, p. 347; Savarésy, pp. 270–7, 288–9; Madrid, pp. 6, 25, 31; Frost, xiii. 30, 35; Comrie, p. 176; Ralph, pp. 66, 75; Rufz, pp. 14, 15; Bally, p. 228; Dariste, p. 164; J. Clark, p. 15; Chisholm, i. 161; Rouppe, p. 306, 410; Davidson, Repos., i. 166; Hunter, pp. 64–69–71; Moseley, pp. 46–49, 437; Blair, p. 75.

epidemics and among different subjects during the same season, the period of excitement being, in general, in inverse ratio to the violence of the attack. But, on a review of all that has been said on the subject, we cannot err greatly in fixing the period of the change in question—which varies from one to five days—at seventy-two hours after the onset of the attack. Be this as it may, at this point, as Dr. R. Jackson remarks, fever, as a progressive process, terminates. The pulse, in the majority of cases, becomes slower and slower, and depressed in point of strength and volume. The uninterrupted and progressive depression occurs also in cases in which the pulse was, at the onset, weak and slow; though in some instances it becomes, in the progress of the disease, more accelerated as, through the effect of art, or the efforts of nature, reaction occurs. “From a calm and uniform tenor, it ordinarily retires silently from the surface and extremities of the body, loses force and expansion throughout, intermits, and finally ceases.”¹ In some instances, though sinking in force, it continues rather quick—in others, again, it increases in frequency towards the close of the attack, and has been known as high as 120 and 130 strokes in a minute, and generally irregular and intermittent.² Dr. Rush states that he met with several cases in which it was full and active, and even tense in the last hours of life (iii. 57). Cases of this kind have been occasionally noticed everywhere. In 1854, among the 254 cases treated at the Roper Hospital, the pulse was full in 26, and strong in 7, in the third stage. In the second stage it was full in 53, and strong in 9. These peculiarities are properly accounted for by the violence of the local determinations, resulting after the yellow fever symptoms proper had passed off.³

The conclusion to be drawn from the peculiar condition of the pulse is, that the yellow fever is not a disease in which the arterial system is necessarily implicated; since the most alarming symptoms, and even death, ensue, when the pulse throughout remains unaltered, or but little changed from the standard of health. We infer from this, also, that the cause of the disease is of a toxical nature, for a like unalteration of the pulse is noticed in other morbid states resulting from the deadly operation of kindred agencies; while the retardation of the pulse is evidently the effect of an altered condition of the blood resulting from the action on the latter of the poison in question.

The reader must perceive, from what precedes, that the pulse is deceptive in the yellow fever in both a prognostical and practical point of view; for it is often found little if at all altered throughout, in cases where the other symptoms indicate a state of great danger, or even portend the approach of death; and in most instances, whatever may have been its condition at the outset, it resumes, in a day or two, its natural standard, and might, if considered with-

¹ Jackson, Sketch, i. 110.

² Ibid., Treat., p. 263; Moseley, p. 438; Bally, p. 295; Madrid, p. 27; N. O. Rep., 1839, p. 333; Tully, p. 297; Townsend, p. 158.

³ Wragg, Charleston J., x. 74.

out reference to other symptoms, lead to a favourable prognosis, when, on the contrary, the disease is progressing to a fatal issue; while in other cases the pulse is found much altered from its natural condition, though nothing else is calculated to create alarm, and the issue proves the correctness of the prognosis. Nor is it less certain that the pulse does not afford us a sure guide in the management of the disease; for it is natural or depressed when active means are called for, and often found useful. In other instances, the same natural pulse is associated with a formidable train of symptoms which forbid the use of the active means alluded to, and call for a contrary treatment, or no treatment at all; and again, in very many instances, it would be unsafe to predicate an active depletory and antiphlogistic plan on the existence, in the stage of reaction, of a full and active pulse; inasmuch as this condition usually subsides after a short period without the aid of evacuant means; and the use of these, if carried to the extent which a pulse of that kind calls for in other diseases, might, by depressing too much the powers of life, give rise to irremediable mischief.

So far, little has been done to ascertain the condition of the heart's action in the yellow fever. Dr. Pennell, who saw the disease on a large scale, at Rio Janeiro, during the epidemic of 1850, and whose attention was early called to the subject, and availed himself of the ample opportunities within his reach, remarks that, upon auscultation, the heart, in an early stage, is often found to be beating violently, but communicates very little impulse to the ribs; suggesting the idea of a small, distant heart, impetuously acting. The sounds of the heart's action are either very much subdued, or one or both are entirely lost. "In other instances, a species of rumbling or churning sound accompanies the disappearing of the natural sounds. Both sounds become less distinct as the disease progresses; but it is yet doubtful whether either one is always and regularly lost before the other. In some instances, the first, and in others, the second seems to be that which was first affected. It is generally the second sound which is first and most seriously affected."¹ These phenomena are supposed to indicate the existence of certain pathological changes, and furnish prognostical indications which will be referred to in their appropriate places.

¹ Med.-Chir. Trans., xxxvi. 247, 248.

CHAPTER VII.

CIRCULATORY SYSTEM CONTINUED.

HEMORRHAGES.—Hemorrhages occupy a conspicuous place in the list of the symptoms of the yellow fever. Though not peculiar to that form of febrile diseases, they constitute one of its main characteristics. They take place in many mild or moderate cases; are absent in but few of the violent, malignant, and fatal ones; and issue from a greater variety of parts than is found in most other complaints. Hemorrhages, according as the blood escapes from or remains in the substance of the parts affected, may, for purposes of description, be divided into external and internal. I shall examine these separately, passing briefly in review the various subdivisions of which they are each susceptible. External hemorrhages, among which we must include only those in which the blood, at the time of its exit, retains those qualities by which its true nature can be easily recognized, without the possibility of doubt, and from among which we must consequently exclude the black vomit and dark intestinal dejections, the matter of which presents appearances somewhat foreign from that of ordinary blood, and has given rise to much discussion—external hemorrhages, I say, proceed from the skin, the lining membrane of the nose, gums, tongue, mouth, from the conjunctivæ, ears, fauces, œsophagus, stomach, large intestines, and bladder.

The frequency of hemorrhages in the yellow fever—the tendency to effusion, especially during the latter stage of the disease, from the several outlets of the body, and through textures and into parts which, in the natural state and in diseases generally, do not afford passage to blood—must depend on a general cause; and that cause no one acquainted with the yellow fever will fail to detect in the particular alteration of the blood, upon which I have already dwelt in detail, and which, by the modifications it imparts to the texture of the capillary vessels, renders them more apt to allow an exit to their contents, or to promote the effusion of the latter in the substance of certain tissues.

The hemorrhagic tendency is, in some seasons, manifested even in individuals who, though under the influence of the efficient cause, are not, properly speaking, affected with the disease, and who otherwise appear to be in the enjoyment of good health. Many of these bleed freely from the nose or other parts. Such was the case here in 1794;¹ and such has been observed at other times and in other places. The tendency may even be said to be exhibited after death; for, in many cases, blood continues, after the cessation of life, to be discharged from the nose, mouth, bowels, &c.²

¹ Rush, iii. 204.

² Ib., iii. 89; Stone, N. O. Journ., p. 182; Levacher, p. 83.

As may readily be inferred from the general description of the fever given in a preceding chapter, and from a consideration of the changes which take place in the condition of both fluids and solids during the progress of the disease, hemorrhages are not equally frequent at all periods. Occurring sometimes in the first stage, they show themselves more generally in the second and last, or that of collapse. In the absence of statistical details illustrative of this statement relative to the fever in this city, I borrow the following fact from a recent publication by my friend Dr. Wragg, of Charleston. In 86 cases of hemorrhage which occurred among the patients treated in the Roper Hospital, during the sickly season of 1854, 10 occurred in the first stage, 43 in the second, and 33 in the last.¹

The exudation of blood which proceeds from some of the outlets specified is, under certain circumstances, the result of an active congestion, or even of an inflammatory irritation of the parts affected. This is particularly the case when it occurs at an early period of the attack, or during or at the close of the first stage; for it is then associated with symptoms of general arterial excitement—is attended often with, or preceded by, evident marks of local irritation, to which it affords relief—or, succeeding immediately to such a condition, is often followed by recovery; all of which would appear incompatible with the idea of capillary atony and relaxation. But, although such is the correct view as regards some of the phenomena mentioned, and although convinced that many of what are regarded as passive hemorrhages are really of a different character, I cannot see any reason for coinciding with those who deny the existence of any other than active hemorrhages in the yellow fever, and maintain that the flow of blood is always—at whatever period of the disease it may occur—the effect of irritation and inflammation.² So far from subscribing to this ultra Broussaian doctrine, we cannot err in considering the sanguineous exudations which take place—in the latter stages particularly—as of a passive character. Whatever may have been the condition of the parts prior to the occurrence, the vessels evidently afford passage to their contents from an atonic or relaxed state of their texture; they continue to discharge those contents after death, when, surely, irritation does not exist; they are associated with symptoms indicative of anything rather than active irritation—a slow, filiform, and often scarcely perceptible pulse, cold extremities, and cold and viscous sweats; they occur in parts in which no irritation can be discovered to exist or to have existed; and the effused blood is of a character the very reverse of that existing in active hemorrhage.

The tendency to hemorrhage in yellow fever, though forming a noted characteristic of the disease everywhere, is not equally conspicuous in all places and at all times. While at Grenada, in 1793, hemorrhages occurred much oftener and more profusely, and were attended with more dangerous consequences than in any other disease—"the scurvy, perhaps, excepted"³—they were but seldom observed at Gibraltar in 1828, at least according to Louis,⁴

¹ Charleston Journ., x. 79.

² Catel, p. 11; *Ib.*, Ann. Mar., 1844, iv. 226.

³ Chisholm, i. 166.

⁴ Pp. 256–258.

who, apparently unaware of the results obtained in other epidemics, wonders how authors could have thought of classing the yellow fever among complaints of an hemorrhagic character. Hemorrhages were not very common in this city in 1853, while they were very much so, during the same season, in New Orleans. (*Fenner*, p. 40.) Dr. John Davy remarks that endemics have been brought to his notice, by medical officers of long experience in the diseases of the West Indies, in which there has been associated with yellowness of skin little or no tendency to passive hemorrhage.¹

In 1827, 1835, and 1838, the tendency to the hemorrhagic condition in the latter stages at Charleston was greater than usual, "and a very large proportion of the cases bled freely, whether they recovered or died." In 1817 and 1824, it was rare, compared with the number of cases and the mortality.² At Martinique, in 1840,³ and at New Orleans, in 1839,⁴ passive effusions were of less frequent occurrence than in the preceding years. Such has been found to be the case also in this city, where, in 1794, as we learn from Dr. Rush (iii. 204), hemorrhages, though occurring in all the grades of the fever, were less frequent than they had been the year before.

This tendency varies also as to extent at different periods of the same season. Dr. Rufz states that, during the epidemic which prevailed at Martinique in 1838 and 1839, hemorrhages were very common in November and December of the former year, and in May and June of the next—in other words, at the commencement and close of the epidemic—while during the intermediate months, this symptom was rarely encountered.⁵ At Barbadoes, in 1847, patients were less prone to passive effusions at the advanced period of the epidemic than at the commencement.⁶

An equal difference exists as regards each individual hemorrhage, in reference to the influence it may exercise on the issue of the case. In one epidemic, a certain form of hemorrhage will be often encountered which, at another season, seldom shows itself, while others are predominant. At one period, some hemorrhages tend to a favourable issue, which at other times fail more or less completely in that respect, and point to an opposite result. But on these subjects I shall more properly dwell while passing each form in review, as well as in the chapter on prognosis; contenting myself here with the remark that hemorrhages are far from always and necessarily indicating fatal results in the yellow fever, or proving by themselves a source of positive danger to the patient. Influenced by such a consideration, there are not wanting those who regard the hemorrhagic tendency as a sign of less importance than is usually supposed; the discharge producing, as they think, when it shows itself, but little change in the condition of the patient, and failing often to furnish correct indications as to the probable issue of the case.

It may even be affirmed that, under certain circumstances, hemorrhages not

¹ Notes to Blair, p. 67.

² Bell's Eclectic Journal, iv. 110.

³ Rufz, Report on, by Chervin, p. 53; Examiner, iii. 57, 72, 120.

⁴ Report, p. 160.

⁵ Chervin's Rep., p. 16; see also Examiner, iii. 57.

⁶ John Davy, Edinb. Journ., lxxii. 280.

only fail to aggravate the disease, but may prove—and, indeed, have proved—salutary by mitigating or permanently removing important or distressing symptoms; and that, in some cases, they have done more, and exercised a critical influence, and hastened convalescence.¹ But such effects will usually be found to occur when the hemorrhage takes place at an early period of the attack, and is of the active kind. If we credit Dr. Catel, of Martinique—and his authority in a question like this can scarcely be ignored—the effect must have been obtained frequently among the troops, admitted into the hospital of Fort Royal. “It is usually from the third to the fifth day that the grave symptoms are developed. It would seem as if nature, reacting violently against the inflammation which threatens to disorganize the viscera, seeks, through means of hemorrhages, to destroy the disease which oppresses it.” Those hemorrhages, which Dr. C. holds to be active, are most profuse when blood has not been freely abstracted by artificial means. “If we judge from the results of observation and experience, we will say that these hemorrhages are critical, and consequently salutary every time the blood flows externally. We have seen individuals lose as much as three litres (or quarts) by the tongue only, and four litres by the nose. These men recovered.”²

It may be doubted whether such things occur often elsewhere; and though hemorrhages undoubtedly occasionally prove salutary, and even critical, we are justified in the belief that, in instances such as M. Catel has mentioned, and many others in which they have been thought to exercise the aforesaid influence, the individuals so affected have recovered in spite, and not through the effect, of the loss of blood they have experienced. Be this, however, as it may, at an advanced stage of the disease, when the effusion assumes the passive character, and is associated with other symptoms of grave import, they generally cease to act in that beneficial way, affording no, or only transient, relief to existing symptoms, and tending in no way to bring about a favourable issue. It may be remarked, however, that, even under such unpromising circumstances, they do not constitute an insuperable obstacle to recovery.³ In some epidemics, hemorrhages have been found less frequently useful than in others, and still less frequently to exercise a critical agency. Such appears to have been the case at Grenada in 1793, where, according to Chisholm, the effusion of blood was never critical, nor in any instance permanently relieved the headache, &c. “I have sometimes been induced to think,” says Dr. C., and his experience coincides with that of Dr. Evans, of St. Lucie, &c. (p. 140), “that it had benefited the patient, by his declaring that the headache had abated in consequence of it; but cold, clammy sweats, an almost imperceptible pulse, and delirium or coma supervening soon after, evinced the imperfect state of the patient’s feelings, and the fallacy of the prognostic” (i. 66).

¹ Wragg, *Charleston Journ.*, x. 79; Lewis, *N. O. Journ.*, i. 300; Cooke, *N. O. Journ.*, x. 643; Fenner (1853), p. 50.

² *Ann. Mar.* 1844, iv. 226.

³ Rush, iii. 149; Warren, p. 19; Harrison, p. 133; Jackson, p. 60.

1. *Skin*.—An exudation of blood from the skin, without abrasion of the surface—though, so far as I am aware, not observed in this city—has not unfrequently been noticed in the yellow fever of other places. Lining remarks, that during the visitations of the disease at Charleston in 1739 and 1745, “there was one or two instances of a hemorrhage from the skin, without any apparent puncture or loss of any part of the scarf skin.”¹ Moultrie, in like manner, noticed such hemorrhages as occurring from the surface of the face, neck, and chest (pp. 4, 7); and so also did Thomas, in New Orleans (p. 42), and Caisergues (p. 170), Berthe (p. 88), and Gillkrest (ii. 271), in Europe. In the West Indies, Labat, in alluding to one of the first recorded epidemics, that of Martinique in 1688, says: “Often there supervened a flow (*débordement*) of blood from all the outlets of the body, even from the pores.”² The same symptom was subsequently noticed by Bourgeois,³ Desportes (i. 198), Bruce (in *Lind*, p. 279), Nasmyth (in *Lind on Seamen*, p. 55), Makittrick (p. 94), Hillary (p. 151), Moreau de St. Méry (i. 702); and though not observed by Rochoux during the period of his residence at Guadaloupe (p. 331), it has been noticed in our town times, and shortly before, in every part of the West Indian Islands, and among these at Martinique; where, as we learn from Savarésy, it is known under the popular appellation of blood-sweat (*sueur de sang*).⁴

The blood, in these cases, issues from the forehead and arm-pits (*Nasmyth*); face, neck, chest (*Moultrie*); from under the nails (*Gillkrest*, ii. 271), and from the scrotum.⁵

As may be presumed, the symptom in question occurs only in the closing stages of the disease; seldom in any but the most malignant and fatal forms of the latter, and is always a sign of portentous character—the harbinger of approaching dissolution. It is generally of restricted locality and of slow progress, the blood oozing gradually, and in a thin sheet, from the minute capillaries.

2. *Nose*.—A flow of blood from the nostrils, varying as to the quantity discharged, and the period at which it occurs, is a much more usual symptom in yellow fever than the preceding, or perhaps than any other form of hemorrhage. From the frequency of its occurrence, as well as from the influence it may exercise on the issue of the disease, or the light it may throw on the probable result of the case, epistaxis deserves the greatest attention. It has been observed everywhere—in this country, in Europe, and in hot climates—to constitute a main element in the symptomatology of the disease. In this city, from the earliest period to the present, its frequency and tendencies have been pointed out and dwelled upon by Currie (p. 22), Rush (iii. 57–204), Caldwell (p. 85), Deveze (p. 26), Jackson (p. 60), Cathrall (pp. 25–40), Nassy (22).

¹ Op. cit., ii. 422.

² Op. cit., i. 73.

³ Voy. Intéressans dans Diff. Col. Françaises, p. 433.

⁴ Chisholm, i. 166; Rufz, Examiner, iii. 57; Bally, p. 236; H. Ferguson, Recoll., p. 146; Savarésy, p. 274; Finlay, p. 21; Hayne, Charleston Journ., vii. 3, 4.

⁵ New Orleans Journ., vii. 43; Barton, Fever of New Orleans in 1833, p. 20; Fenner, Fever of same city in 1853, pp. 49, 50.

In other cities of this continent, from Texas to Boston—writers on the yellow fever¹ have found this form of hemorrhage to be of equally frequent occurrence. So also with those in Europe;² and there is scarcely a writer on the fever of tropical climates who has not dwelt, more or less, on the same subject.³

Epistaxis occurs sometimes during, or at the close of the first stage of the disease;⁴ but more frequently—indeed almost universally—it is a symptom of the last stage, occurring towards the fifth or sixth day, and continuing to the end. In the former case, when the energies of the system have not been exhausted, and when the excitement has been great, this form of hemorrhage may be hailed as a favourable omen, leading, as it often does, to salutary results, as evinced by a gradual, sometimes rapid diminution of the more alarming symptoms; and if, in other instances, such an amelioration fails, and death succeeds, the issue may be ascribed rather to the violence of the disease, over which the salutary effort of nature could exercise no control, than to the effusion of blood. In those cases, on the contrary, when the hemorrhage take places at a later period, it is more rarely followed by an amendment of the symptoms; the disease usually pursuing its course to a fatal issue uninfluenced by the tardy effort; while the effusion which continues to, or occurs towards the close of the disease, assumes a passive character, and consists of altered blood, and, being associated with a relaxed condition of the capillary vessels, and a prostration of the vital powers generally, is seldom very copious, but the result of a gradual filtration. Under such circumstances it is rarely if ever followed by recovery, and may even tend to hasten the fatal issue, if, as sometimes occurs, it is very profuse.

Nor does the difference of result rest solely on the period of the disease at which epistaxis occurs. Much will also be found to depend on the mode of the effusion; for experience has proved that in order to be beneficial, or not to prove the forerunner of a fatal result, the flow of blood must, even at the

¹ Dalmas, p. 8; Michel, *Charleston Journal*, v. 745; Lining, ii. 422; Waring, p. 46; Girardin, p. 23; Thomas, p. 84; Archer, p. 69; Gros, pp. 9, 11; Moultrie, p. 4; Davidge, p. 103; Harrison, p. 133; A. Hosack, p. 17; Townsend, p. 160; Shecut, p. 121; Fenner, 1853, pp. 49, 50; Kelly, p. 350; Valentin, p. 170; Munson, p. 180; Seaman, *Fever of 1796*, p. 8; E. H. Smith, p. 116; Ashbel Smith, *Tr. of Acad. of Med. of N. Y.*, i. 61, 62; Wragg, *Charleston Journ.*, x. 79.

² Chirac, i. 53; Gillkrest, p. 271; Pym, pp. 233–4; Audouard, p. 58; O'Halloran, *Fever of S. of Spain*, pp. 85–130; Blin, p. 7; Pariset, pp. 376–429; Amiel (in Johnson), p. 264; Caisergues, pp. 169–179; Fellowes, p. 55; Berthe, p. 87; Robert, *Guide S.*, i. 253; Louis, pp. 256–7; Vance (in Pym), p. 127; Rochoux, p. 493.

³ Bruce, p. 279; Desportes, i. 198; Moseley, p. 428; Holliday, p. 10; Evans, p. 257; Caillot, pp. 17–19; Bancroft, p. 34; Hillary, p. 150; Lempriere, ii. 65–84; Dickinson, 126–129; Arnold, p. 10; Warren, p. 15; Ferguson, p. 146; Rufz, p. 15; Bally, pp. 230–5; Dariste, 160–5; Pugnet, p. 357; Gillespie, p. 41; McArthur, p. 347; Savarésy, p. 273; Chisholm, i. 166; Imray, liii. 79, 81; Gilbert, p. 66; J. Clark, p. 7; Leblond, p. 104; Rochoux, p. 331; Nasmyth (in Lind on Seamen,) p. 55; Finlay, p. 21; Bourgeois, p. 433.

⁴ Cathrall, Girardin, Caillot, Gros, Moultrie, Currie, Dickinson, Harrison, Audouard, Dariste, Jackson, Desportes, Gilbert.

most propitious moment, be somewhat abundant; and that, in this, as indeed in other acute diseases,¹ a trifling effusion of a few drops, repeated several times a day, is not only found unproductive of a favourable change, but the harbinger of immediate danger. Not unfrequently, however, the patient, who may perchance lose, as seen above, with impunity, a large amount of blood through the outlet in question, is thereby rapidly reduced, and the fatal event is accelerated by the great amount of the fluid discharged.

Hemorrhage from the nostril is generally increased by the slightest pressure or friction. As may be inferred from what was said in reference to hemorrhages generally, epistaxis occurs more frequently in some epidemics than in others. It was very common at Dominica in 1838,² and at Pomergue near Marseilles in 1822.³

3. *Gums, Tongue, and Mouth.*—Exudation of blood from the lining membrane of the mouth, gums, and tongue, separately or conjoined, though not as generally observed as the preceding form of hemorrhage, is, nevertheless, frequently encountered in the yellow fever, whether of temperate or tropical regions. In the various epidemics of this city, it has not been a very unusual symptom, as recorded by Rush (iii. 57), Currie (p. 28), Cathrall (p. 31), Deveze (p. 26), Barnwell (pp. 386–7), Caldwell (p. 85), and S. Jackson (pp. 56–60).

In other cities of this country, it has not been less usually noticed.⁴ At Barcelona, in 1821, according to Rochoux (p. 495), probably one-fourth part, and, according to Pariset, one-sixth or one-fifth, of those attacked with the disease, suffered from the phenomenon in question; and though by other writers on this and the other epidemics of Europe, nothing is said of its relative frequency, we may infer from their statements that, if it has not always been as frequent as is mentioned by Rochoux and Pariset, it is sufficiently so to shield them from the charge of exaggeration.⁵ Certain it is, that, in tropical regions, it has been very commonly noticed at every repetition of the disease.⁶

¹ Hippocrates, De Morb. Vulg., lib. i. Coacæ Prænot. No. 57, 179.

² Imray, liii. 83.

³ Robert, i. 253.

⁴ Moultrie, pp. 4, 7; Lining, ii. 422; E. H. Smith, p. 16; Gros, p. 11; Ashbel Smith, Tr. of Ac. of Med. of N. Y. i. 61, 62; Valentin, p. 170; Waring, p. 46; Dickson, pp. 350–413; Munson, p. 180; Townsend, pp. 160, 1–178; Shecut, p. 121; Irvine, p. 29; A. Hosack, p. 17; Kelly, p. 380; Wurdeman, Am. Journ., N. S., ix. 52; Archer, p. 68; Pascalis, Marseilles Journ., v. 140–1; Harrison, p. 133; New Orleans Journ., vii. 43; Thomas, p. 84; Tully, p. 299; C. Drake, xxi. 135; Revere, p. 226; N. O. 1820, p. 10; Ib., 1839, p. 333; Seamen, 1795, p. 8; Fenner, 1853, pp. 49, 50; Stone (Woodville), p. 182; Wragg, Charleston Journ., x. 79.

⁵ Arejula, p. 160; Jackson, pp. 76–100; Gillkrest, p. 271; Fellowes, p. 62; Velasquez, p. 12; Palloni, p. 5; Vance (in Pym), p. 127; Gilpin, v. 322; Berthe, p. 87; Proudfoot, xxvii. 250; Caisergues, p. 169; Robert, i. 253; Louis, p. 256; Audouard, p. 68; O'Halloran, p. 85; Ib., Fever of S. of Spain, p. 130.

⁶ Warren, p. 15; Holliday, p. 10; Dariste, pp. 128–165; Lempriere, ii. 65–84; Gillespie, p. 41; Pugnet, p. 357; Imray, liii. 81; McArthur, p. 347; Savarésy, pp. 273–4; Rochoux, p. 334; Ib., F. I., p. 151; Moseley, p. 438; Caillot, p. 19; Bancroft, p. 14; Pym, p. 233; Dickinson, p. 133; Hillary, p. 151; Chisholm, i. 166; Ferguson, p. 146;

Everywhere hemorrhages from some portion of the lips have been found, occasionally, to be associated with those from the gums or internal lining of the mouth, and have been noticed in our early and later epidemics.¹

Hemorrhage from the parts mentioned, like that from the pituitary membrane, takes place sometimes at an early, though more usually at the closing stage of the disease. Though a symptom of serious import, it does not accompany fatal cases only, and must not be viewed, apart from other symptoms, as indicative of inevitable dissolution, especially when it occurs early.

In these cases it continues, sometimes at intervals, to the close of the disease, and even, according to Pariset (p. 413) and other observers, during convalescence, the progress of which it does not impede. Assuming, at first, the character of a slow exhalation, by which the saliva is simply tinged of a reddish hue; the quantity effused gradually increases until it becomes very considerable. The blood thus discharged is more or less pure; but, in consequence of being retained for some time in the mouth, and then mixed with air and saliva, it becomes slightly decomposed, and acquires the fetid odour noticed in most cases.² This form of hemorrhage, when it occurs at an early period of the disease, has been said to prove innocuous, salutary, and even critical.³ Audouard even affirms that the greater number of those who suffered from it at Barcelona recovered (p. 69). But such favourable results are not generally obtained. Even when the case terminates promptly and soon after the appearance of the hemorrhage, the same amendment occurring in the same way when no effusion takes place, and the latter being generally small in quantity and occurring, too, slowly, we are scarcely justified in attributing the relief to it, and more particularly in regarding it as possessed of critical powers. More frequently the exudation is not found to exercise any influence, for good or evil, in the progress or issue of the case, whatever be the period at which it appears. It continues from five to eight days, is slow in its progress, and ceases, in cases of recovery, as the strength is restored; while, in fatal cases, the symptoms are not amended by the discharge of the blood, and, in some instances, indeed, death appears to be hastened by large quantities effused.

Though usually possessing the little importance mentioned, hemorrhage from the mouth deserves the attention of the physician, inasmuch as it is often the prelude of sanguineous effusions from internal organs, and indicates, besides, the commencement of the morbid change in the blood which forms one of the characteristic marks of the yellow fever. In a few instances, the he-

Rufz, p. 15; Bally, pp. 205-236; Furlong, p. 290; Belcher, p. 251; Dancer, p. 83; Levacher, p. 74; Frost, xiii. 32-6; Maher, pp. 849-853; Ralph, pp. 69, 71, 74; Catel, p. 11; Blair, p. 64; Leblond, p. 104; J. Davy, Notes to Blair, pp. 30, 64, 5; Nasmyth, in Lind on Seamen, p. 45, note; Bruce, p. 278; Lefort, de la Saignée, p. 569; Vatable, p. 346; R. Jackson, Treat., pp. 260, 1; Sketch, i. 111; Barrington, xii. 312.

¹ Valentin, p. 170; Clark, p. 13; Gros, p. 11; Bally, p. 235; S. Jackson, pp. 56-60; Deveze, p. 26.

² Rochoux, p. 334.

³ Kelly, xiv. 380; Periodico, &c., p. 266; Catel, An. Mar. 1844. p. 226; Rufz, p. 16; Examiner, iii. 57.

morrhage from the gums or tongue has, combined with slowness of the pulse, constituted the only symptom by which the nature of the disease could be ascertained.¹ Like hemorrhage from other parts, that from the gums, mouth, or tongue, varies in point of frequency in different epidemics. While, according to Rochoux, it occurred in one-fourth of the patients, and, according to Pariset, in one-sixth or one-fifth, at Barcelona, in 1821, Louis informs us that, of thirty-five patients, not dissected by him (and Trousseau), one had a hemorrhage of the tongue, and four or five lost blood from other parts of the mouth. In the cases they dissected, there does not appear to have been effusions of this kind. Of the patients who recovered, and an account of whose symptoms they noted, one had hemorrhage from the tongue and inside of the gums. In thirty-three cases communicated to them, four had hemorrhage from the tongue. Seven out of twenty-three soldiers lost blood by the mouth. He adds that, "in the patients who recovered, as in those who died, and with whose history they have been made acquainted, the hemorrhages were limited to a very small number of organs." There was none from the mouth (pp. 256-8). It may be remarked that the number of individuals who recover, after experiencing hemorrhage from the gums, varies in different epidemics. In that of Dominica, in 1841, more recovered than had done three years before.²

4. *Fauces—Pharynx—Œsophagus.*—Hemorrhage from these parts not unfrequently occurs in the latter stages of the disease, and sometimes at an early period. It has been noticed in this city by Currie (p. 28), as also elsewhere by Archer (p. 67), E. H. Smith (p. 116), Caisergues (p. 179), Palloni (p. 5), Pariset (p. 412), Ralph (p. 74), Rochoux (p. 335), Blair (p. 30), Dickson (p. 350), Leblond (p. 104), Irvine (p. 29), Proudfoot (xxvii. 250).

This form of hemorrhage, when it occurs during the early part of the disease, exercises little influence over the issue of the case—neither aggravating nor ameliorating the condition of the patient. When, however, it occurs in an advanced stage, and is accompanied—as is generally the case—with a sense of ardor and constriction in the part affected, and extending down towards the stomach, this symptom acquires great importance, and must be viewed as indicative of imminent danger. It is preceded and announced, in most cases, by pain and heat in the fauces and about the neck; and, on examination, the part will often be found to be red and engorged. But at whatever time hemorrhage of the kind may occur, it is entitled to our regard in a diagnostical and prognostical point of view, and may, if not correctly detected, lead to erroneous conclusions. The blood is not always discharged from the mouth in a pure state; but whether so or otherwise, it collects in the mouth, or finds its way into the stomach, whence it is thrown off after having assumed a dark appearance, and suffered partial decomposition. Under these circumstances, blood effused from the parts mentioned may be and has often been erroneously thought to proceed from the capillaries of the stomach; it has thus, consequently, been mistaken for the black vomit.³

¹ Pariset, p. 414.

² Imray, Edinb. Journ., liii. 83; Ib., lxiv. 329.

³ Rochoux, p. 335.

5. *Eye*.—In some, but not many cases, and only at the close of the disease, blood flows in small quantity from the canthus of one or both eyes. This has been observed occasionally in this city (*Rush*, iii. 57), and is taken notice of by writers on the fever of other parts of this country, as well as of Europe and tropical regions.¹ The effusion consists either of a sero-sanguineous fluid, or of blood more or less pure. In some cases it succeeds to inflammatory irritation of the conjunctiva, or occurs without more than the congestion ordinarily observed in that part in the disease. It is never critical, nor beneficial in its effects; never sufficiently profuse to aggravate the symptoms, but usually indicates considerable danger.

6. *Ears*.—An hemorrhagic effusion from the ears has occasionally been observed. Dr. Rush (iii. 57) enumerates it among the symptoms of the fever in 1793; and it is mentioned as not an unusual occurrence in this country, in Europe, and the West Indies.²

Like hemorrhage from the eyes and other parts, the one in question is sometimes seen very rarely, and at others, again, fails completely during the course of a wide-spreading epidemic. Dr. Rufz did not see or hear of a case during the prevalence of the disease at Martinique in 1839.³ Neither was it observed by Louis at Gibraltar in 1828 (pp. 256, 8).

It usually occurs at an advanced period of the disease; and so far as I am able to say, no one, with the exception of Desportes, has regarded it in the light of a critical discharge, and as exercising a salutary influence over the disease, while it cannot, any more than the preceding, entail any addition to the danger of the case, owing to the limited quantity of blood effused.

7. *Stomach*.—In not a few cases, blood is effused from the internal surface of the stomach. Being voided by vomiting in a greater or less degree of purity, and thereby giving rise to real hæmatemesis, it must be distinguished, for the present, from the discharge of black matter which constitutes one of the main characteristics of the yellow fever. This form of hemorrhage has been noticed in this city by Dr. Rush (iii. 57) and others, and is mentioned by almost every writer on the yellow fever of temperate and tropical climates.⁴

¹ A. Hosack, p. 17; Gros, p. 11; Lining, ii. 422; Hayne, Charleston, J., vii. 4; Archer, p. 68; Harrison, p. 133; Amiel (in Johnson), p. 264; Fellowes, p. 55; Pym, p. 233; Bruce, p. 279; Moseley, p. 438; Savarésy, p. 274; Hume, p. 199; Dickinson, p. 136; Chisholm, i. 166; Ferguson, p. 146; Catel, p. 11; Bally, p. 236; Dariste, p. 165; Makittrick, p. 94; Nasmyth, op. cit., p. 45; Levacher, p. 74; Rochoux, pp. 334–493; Holliday, p. 10; Dickson, p. 350; Michel, Charleston J., v. 745; Leblond, p. 104.

² Lining, ii. 422; Waring, p. 46; Townsend, p. 161; A. Hosack, p. 17; Harrison, p. 133; Hume, p. 199; Caisergues, pp. 169–179; Pym, p. 233; Palloni, p. 5; Vance, p. 127; Cayenne in 1850, p. 174; Bruce, p. 279; Desportes, i. 198; Ferguson, p. 146; Chisholm, i. 166; Dickinson, p. 136; Holliday, p. 10; Levacher, p. 74; Blair, p. 65.

³ Chevin's Rept., p. 16; Exam. iii. 57.

⁴ Lining, ii. 422; Girardin, p. 34; Wragg, p. 79; Davidge, p. 103; Dickson, p. 350; E. H. Smith, p. 116; Seaman, p. 8; Waring, p. 46; Ashbel Smith, Tr. of N. Y. Ac., i. 61; Audouard, p. 59; Pariset, pp. 418, 419; Blin, p. 7; Caillot, p. 19; Evans, p. 240; Dickinson, p. 136; Ralph, pp. 68, 74; Blane, p. 446; Gillespie, p. 41; Bally, pp. 237, 239; Jackson, i. 76, 94, 111; Dariste, p. 165; Rochoux, p. 260; Maher, p. 842.

In some instances the blood is ejected in a fluid state; at others, wholly or partially coagulated. Generally occurring in the latter stage of the concentrated form of the disease, hemorrhage is seldom if ever beneficial, or even free from danger, and must, on the whole, be viewed as a symptom of bad omen. It often precedes the discharge of real black vomit; but, in some cases, alternates with, or succeeds to it—the quantity discharged varying in different cases. This form of hemorrhage is not unfrequently found associated with a sensation of roughness and dryness of the tongue, as well as with considerable thirst, and a nauseous and sweetish taste in the mouth. (*Jackson*, i. 111.) The quantity of blood thus discharged is often prodigiously great—sufficiently so to fill a large basin.

8. *Bowels and Anus*.—As associated often with the preceding, and in many cases occurring independently of it, hemorrhage from some portion of the intestinal canal deserves attention. It is, perhaps, more common than hæmatemesis; for, while the patient seldom voids blood by the stomach without voiding it also by the bowels, it not unfrequently happens that, in cases unattended with hæmatemesis, the discharge of black vomit is associated with true intestinal hemorrhage. This symptom has, indeed, been pointed out everywhere as one of usual occurrence in the disease under consideration, and is almost characteristic of it.¹ Discharges of the kind are sometimes very profuse, amounting, in some cases, to not less than several pounds; the stools having been, on these occasions, entirely composed of the pure fluid. These profuse hemorrhages never fail to prove detrimental, hastening often the fatal issue of the case; while the same effusion, however properly restricted it may be as to quantity, or occurring in the early period of the disease, seldom if ever produces a salutary effect. Desportes, however, whilst admitting that such is the case ordinarily (i. 167), appears to have occasionally found hemorrhage from the anus to exercise a critical agency (i. 198).

9. *Genito-Urinary Organs*.—Hemorrhages from the bladder, and parts connected with this organ, have been noticed by Dr. Rush (iii. 57) as occurring in 1793, as also by numerous writers.² The effusion is either in small quantity, and sufficient only to tinge the urine with blood, or it is profuse, and consists of pure blood. In one case, referred to by Rochoux, the quantity voided from the bladder amounted to near a pound (p. 336). The occur-

¹ Rush, iii. 57, 204; Deveze, p. 24; Currie, pp. 26, 28; Caldwell, p. 85; Gros, p. 11; Harrison, p. 133; Waring, p. 46; Valentin, p. 170; Palloni, p. 6; Amiel, p. 264; O'Halloran, pp. 85, 130; Fellowes, p. 55; Jackson (Spain) p. 103; Caisergues, pp. 169, 179; Blin, p. 8; Blane, p. 446; Davidson, p. 103; Pugnet, p. 357; McArthur, p. 347; Savarésy, p. 274; Chisholm, i. 166; Bally, pp. 230, 235, 6; Evans, p. 257; Levacher, p. 74; Caillot, p. 19; Ralph, p. 74; Gilbert, p. 66; R. Jackson, i. 76, 94, 111; Moultrie, p. 4; Thomas, p. 84; Stone (Woodville), p. 182; Dickson, p. 350; Wurdeman, Am. J., N. S., ix. 52; Michel, Charleston J., v. 745; A. Smith, Tr. Ac. of Med. of N. Y., i. 61.

² Pariset, p. 424; Lining, ii. 422; Waring, p. 46; O'Halloran, p. 130; Bally, pp. 235, 6; Stone (Woodville), p. 182; Gillespie, p. 41; Savarésy, p. 274; Berthe, p. 88; Evans, 240, 257; Rochoux, p. 336; Desportes, i. 198; Dict. des Sci. Méd., xv. 337; Levacher, p. 72; Michel, Charleston J., v. 745; Dickson, p. 350; Wragg, op. cit., p. 79; Nott, ix. 283.

rence of this hemorrhage must usually be viewed in the light of an unfavourable complication. It depends on the irritation or congestion of the mucous membrane of the parts, a morbid state occurring often without assignable cause, and apparently as the natural result of the general disease; but which in some cases is due to the action of cantharides. It is sometimes associated with a suppression of urine. When unconnected with this deadly symptom, it may prove, and has proved advantageous under particular circumstances, and even exercised a critical influence. (*Desportes*, i. 199.)

This hemorrhage, which in some epidemics is of more or less frequent occurrence, is seldom, or even not at all, observed in others. According to Louis, it was not seen in any of the cases—fatal or otherwise—which he saw himself, or with whose histories he was furnished, at Gibraltar in 1828 (pp. 256, 8).

10. *Uterus*.—Hemorrhage from the uterus or vagina is of not unfrequent occurrence in the yellow fever. It is perhaps more commonly encountered than that from the bladder. In the year 1793, in this city, uterine hemorrhage was, as well as that from the nose, the most frequent during the early part of the epidemic.¹ It has been many times observed in other places.² A distinction appears necessary to be made between uterine hemorrhage in yellow fever and the natural flow of the menses. Women, says Deveze, who were fortunate enough to have their monthly discharge at this period, very generally recovered, though this discharge occurred before the regular time (p. 31). On the other hand, it was observed at Barcelona, in 1821, that every circumstance which occasioned a flow of blood from the uterus—as an accouchement, an abortion, and excessive menstruation, even—proved fatal when it coincided with the yellow fever.³ Palloni is about the only author who has seen a case of abortion, attended with profuse hemorrhage, end in recovery.

11. *Lungs*.—Dr. Rush mentions a case in which hemorrhage took place from the lungs, and was mistaken for common hæmoptysis;⁴ and other writers speak of effusion from the pulmonary membrane.⁵ Louis, on the other hand, states that at Gibraltar, in 1828, no hemorrhage from the air-passage, at least in subjects with whose histories he became acquainted, was observed. Indeed, from the paucity of records relative to this form of hemorrhage in yellow fever, we are justified in suspecting that its occurrence is extremely rare, and may be viewed more frequently in the light of a coincidence than as the effect of the hemorrhagic tendency manifested in other parts; though we can see no reason why it may not occur occasionally there as well as elsewhere.

¹ Rush, iii. 57; Jackson, p. 56.

² Valentin, p. 170; Davidson, p. 103; Berthe, p. 88; Savarésy, p. 274; Bally, p. 230–235; Waring, p. 46; Cooke, N. O. Journ., ix. 642; Pugnet, p. 357; Ralph, p. 74; Palloni, p. 6; Pariset, p. 424; Audouard, p. 64; O'Halloran, p. 130; Deveze, p. 31; Levacher, p. 74; Seaman, p. 8.

³ Audouard, p. 69; Pariset, p. 425; Rochoux, p. 494.

⁴ Vol. iii. p. 57.

⁵ Girardin, p. 34; Pariset, p. 447; Wragg, op. cit., p. 79.

12. *Wounds, Sores, and Denuded Surfaces.*—The tendency of the blood to transude through surfaces unaltered in their texture, being such as I have described, we may readily expect to find it to escape with greater facility when those surfaces are in any way abraded. Hence, Dr. Rush found, in 1793, that “many bled from the orifices which had been made by bleeding, several days after they appeared to have been healed, and some from wounds which had been made in veins, in unsuccessful attempts to draw blood.” (Vol. iii. p. 57.) Audouard noticed kindred phenomena at Barcelona in 1821 (p. 69), and Gilbert (p. 66) and others have done the same in the West Indies.¹ Blood has also been found to ooze for days from imperfectly healed leech-bites, easily controlled by compression, but recommencing as soon as this compression is withdrawn.² It has not unfrequently been found, also, that blood exudes from blistered surfaces;³ from old and accidental sores or carbuncles;⁴ from the wounds produced by scarifications or cuts;⁵ from ordinary wounds;⁶ from cicatrices,⁷ bruises,⁸ and excoriations.⁹ Dr. Wragg says: “In one case, the small incision made for evacuating the contents of a thoroughly matured boil continued to bleed so profusely as to weaken the patient seriously, and it could only be arrested by an ingenious application of adhesive plaster coated over with collodium” (p. 80).

In the very large number of instances, these hemorrhages occur in the latter stages of the disease, and produced, as they evidently are, by, or connected with, an impaired vital cohesion in the capillary vessels, and a disintegration of the circulating fluids, must be viewed as events of fearful omen. In some cases, also—as when the blood flows from the unhealed orifice of a vein—the hemorrhage is not only very troublesome, but, occasionally, hastens the fatal issue. (*Rush*, p. 57.) But neither this form of hemorrhage, nor the others, proceeding from leech-bites or denuded surfaces, are necessarily the harbingers of death, for it has been found, in this city, as well as at Barcelona and elsewhere, that recovery takes place, notwithstanding a continued oozing of blood during several days—the hemorrhage ceasing on the approach of convalescence. (*Audouard*, p. 69.)

Internal Hemorrhages.—Blood is not only effused externally from various surfaces, and in the way mentioned, but it is not unfrequently found that, through the instrumentality of the same hemorrhagic tendency, and the same deficiency of vital energy in the capillary vessels and of cohesiveness in the blood, which promotes the occurrence of these phenomena, the fluid escapes

¹ Deveze, p. 26; E. H. Smith, p. 116; Waring, p. 46.

² Audouard, p. 69; Pariset, &c., p. 233; Louis, p. 256; Rochoux, p. 496; Stone, (Woodville), p. 182; Kelly, p. 379.

³ A Hosack, p. 17; Lining, ii. 422; Wragg, p. 80; Caldwell, p. 85; Monges, p. 61; Louis, p. 256; Stone (Woodville), p. 182; Pariset, p. 287; Rochoux, p. 497; Finlay, p. 21; Waring, p. 46; Wurdeman, *Am. Journ. Med. Sci.*, N. S., ix. 52.

⁴ Caldwell, p. 85; A. Hosack, p. 19; Blair, p. 27.

⁵ Wurdeman, *op. cit.*, p. 52; Stone (Woodville), p. 182; Wragg, p. 80.

⁶ Lefoulon, p. 104.

⁷ Nasmyth, p. 55.

⁸ Wragg, p. 80.

⁹ Finlay, p. 21.

from the vessels; but, finding no vent externally, is extravasated in the substance of the tissues or organs. These hemorrhages, which, in contradistinction to the former, are denominated internal, are exhibited in the form of petechial spots, of ecchymoses, and intermuscular or cellular collections.

1. *Petechiæ*.—Considerable doubt exists in the minds of pathologists respecting the existence of real petechiæ in the yellow fever. These imply the existence of a morbid change in the substance of the cutis, with subsequent desquamation after recovery—phenomena which are not found to occur in the spotted eruption of this disease; in a word, petechiæ constitute a peculiar exantheme, and form part of a class of complaints to which, so far as we can perceive, the yellow fever does not belong. This is not the place to enter into an examination of the subject, and I must content myself with remarking that, whatever be thought of the occurrence of true petechiæ in the disease before us, it is undeniable that a flea-bite-looking eruption, due evidently to the simple extravasation of blood in small points, and constituting a form of internal hemorrhage, is observed, in the latter stages of the disease, in the mucous membrane of the stomach, and other parts, as well as under the cuticle. To this subject I shall revert in the next chapter.

2. *Ecchymosis*.—Whatever difference of opinion may exist concerning the hemorrhagic character of petechial spots observed in the yellow fever, there can be none as regards the nature of the ecchymoid blotches which, as has already been stated, occur very generally at the close of the attack; for no one can doubt that they are the result of the effusion of blood, as well under as in the substance of the skin. When the spots are small, the thickness of the extravasation is but limited; under opposite circumstances, the hemorrhage penetrates deeply, and invades the entire substance of the skin.

3. *Subcutaneous and Intermuscular Hemorrhages*.—Blood is not unfrequently found extravasated, in greater or less quantity, in the cellular tissue covering the muscles, and situated between the adjacent muscular fibres. It is generally of a dark colour, decomposed, often in an incipient state of putrefaction, and in all but recent cases has a fetid odour; which, however, must not be confounded with that of gangrene. These hemorrhages have been often noticed in the fever of hot climates, and, notwithstanding the doubts expressed by Dr. Rochoux (p. 498) in relation to this matter, are not uncommon in the yellow fever of temperate regions.¹

The existence of these collections is announced, during life, by acute pain and sometimes swelling of the part affected. But these symptoms are only manifest when the effusion is very large; in other cases, when but little blood is effused, the effects produced being of trifling character, are unobserved,

¹ Pugnet, p. 357; Catel, Ann. Mar., 1844, iv. 227; Ib., Rep. on Fev. of 1838, p. 12; Levacher, p. 74; Gillkrest, ii. 272; Rochoux, p. 338; Furlong, p. 290; Kéraudren, Arch. Gén., xv. 459; Maher, p. 886; Deveze, p. 80; A. Hosack, p. 17; Valentin, p. 170; Michel, op. cit., p. 746; Fellowes, p. 57; Louis, 254; Gillkrest, ii. 272–277; Pariset, p. 341; T. Smith, Edinb. Journ., xxxv. 45; Ashbel Smith, Trans. Acad. of New York, i. 61; Kelly, xiv. 379.

and the condition of the part is only discovered on dissection, or is only suspected by the existence of ecchymoses of uncommon size.

It is well remarked by Rochoux that such hemorrhages constitute lesions of a dangerous character, and might themselves occasion the death of the patient by the derangement they would inevitably produce, were he to survive long after their appearance. In all the cases of the kind Rochoux saw, death was in some measure produced by them (p. 339).

Louis found an effusion of this kind in the parenchyma of the lungs (p. 255). Dr. Michel, of Charleston, saw hemorrhagic collections in the substance of the eye, with destruction of the organ.¹ Kindred collections have been found beneath the peritoneum; in the cellular substance investing the kidneys and renal capsules; between the laminae of the sac of Willis; in the substance of the kidneys, liver, bladder, and in the subcutaneous cellular membrane of every portion of the surface.²

The quantity of blood thus effused varies considerably, from a gill up to quarts.

CHAPTER VIII.

STATE OF THE SKIN.

IN surveying the symptoms of the yellow fever as they manifest themselves on the surface of the body, we are to consider the changes which the skin undergoes in its double capacity as organ of sensation and organ of secretion; as well as various morbid modifications to which it is liable during the progress of the attack. This examination, thus conducted, will embrace an inquiry into the temperature of the surface; 2, the state of the skin in reference to its perspiratory function; 3, the modifications of colour it undergoes; and 4, the diseases which supervene in it during the course of the disease.

A. *Temperature*.—The complaint which forms the subject of our inquiries being, as its name imports, classed among those of a febrile kind, we might, *à priori*, conclude that it is characterized by increased temperature of the skin, inasmuch as this constitutes one of their most usual attributes. Hence, in the foregoing description of the disease, it was stated that, in some of its forms—the inflammatory—the skin, after the coolness or coldness of the opening stage has subsided and reaction has been established, becomes hot and parched, or imparts a burning sensation. That this was not uncommonly the case in our early epidemics we learn from the description of them handed down to us by our predecessors. Dr. Rush states that, in 1793, the skin was preternaturally warm in some cases (iii. 63), and that in 1794, “the burning heat on the skin, called by the ancients *calor mordens*, and from which this fever, in some countries, has derived the name of *causus*,

¹ Op. cit., pp. 7–46.

² A. Smith, op. cit., p. 61.

was more common than the year preceding" (iii. 212). Similar statements are made in reference to the first epidemic by Deveze (p. 22), Currie (p. 21), Cathrall (p. 24). That the same character was not less common in 1797, we learn more particularly from Dr. Currie (on *Bil. Fev.*, p. 219), and that such was the case subsequently we know from personal observation, and the testimony of Ffirth (p. 26), Caldwell (p. 80), S. Jackson (pp. 52, 59, 61).

A like condition of the skin has been over and over again observed in various parts of this country from Boston to Galveston.¹

What is true in reference to the fever in this and other American cities, will be found equally so as regards that of the south of Europe. In the general descriptions of the fever of Leghorn by Palloni, Pasquetti, Moscati, Brignole, Dufour, and others, we are told that the skin was hot, burning, and dry, and was acrid to the touch—*calor ardentissima*—according to the first of these writers.² In the fever which prevailed at Cadiz in 1800, the cold fit was succeeded, according to Caisergues (p. 167), Berthe (p. 84), Blin (p. 6), De Maria (p. 64), by a similar symptom—"an acrid, burning heat, which, according to the patients, was as pungent interiorly as it appeared to be to the touch." Doughty found the skin hot at Cadiz in 1810 (pp. 126, 135, 137, 149). "In Seville, 1819, Velasquez (Pariset, *Observ.*, p. 10) found the surface burningly hot." Pariset says the same thing in reference to the fever of Cadiz of the same year, on the authority of Flores, Gonzales, Coll, Ameller, and Arejula (p. 29). In the epidemic of 1820, Dr. R. Jackson found the heat of the surface "often strong, sometimes ardent, especially at the præcordia" (p. 71). The epidemics of Malaga, Carthagen, Barcelona, and Gibraltar, have furnished many examples of the kind.³

References so numerous, to authorities so reliable, establish not only the correctness of the statement, that the yellow fever of Philadelphia is, if not always, at least often, characterized by a hot skin, but also that on that point it assimilates itself to the fever of other parts of temperate regions, and, by so doing, furnishes an indisputable proof of the erroneousness of those who maintain that in the fevers of those regions the skin never exhibits the condition in question.

While such is the case in this country and Europe, it will be found that the hot burning skin is a common symptom in the fever of tropical regions.⁴

¹ Bayley, p. 96; Pascalis, Marseilles J., v. 144; Dalmas, p. 6; D. Hosack, Practice, p. 388; Alex. Hosack, p. 11; Townsend, p. 146; C. Drake, xxi. 134; S. Brown, p. 12; Revere, iii. 224; Drysdale, i. 29, 132; Davidge, p. 102; Archer, v. 66; Valentin, p. 165; Hill, v. 89; Irvine, p. 29; Dickson, iii. 254; Ib. Eclect. Journ., iv. 119; Munson, p. 179; Seaman, p. 7; Stone, p. 181; Shecut, p. 119; Kelly, p. 377; Merrill, ii. 222; ix. 244; Cartwright, ix. 13; Hogg, i. 412, 416; Girardin, p. 13; Harris, Am. J., xiv. 69; Gros, p. 9; Rept. on Fev. of N. O. in 1819, p. 8; Ib., on Fev. of 1820, p. 8; and 1839, p. 331; Harrison, p. 132; A. Smith, of Galveston, Am. J., xxv. 501; Ticknor, iii. 224, 5.

² Febbre Gialla, p. 4; Caisergues, p. 177; Tommasini, pp. 9, 10, 137; Dufour, iv. 50.

³ Arejula, p. 167; Rayer, p. 21; Pariset, p. 394; Amiel (in Johnson), p. 263; Fellowes, p. 200; Proudfoot, xxvii. 249, 251; Louis, pp. 205, 6; Gillkrest, ii. 270; Audouard, p. 56.

⁴ Poissonnière, p. 50; Bruce (Lind), 278; Hunter, p. 69; R. Jackson, Tr., pp. 254, 280;

From what has thus been adduced, we perceive that the stage of reaction in the yellow fever is everywhere often characterized by increased temperature of the surface. Dr. Hales believed that the heat of the blood in high fevers rose to 136. Boerhaave, from the experiments he made, was led to think that much mischief would accrue from a degree of heat above 106, on the supposition that the serum would thereby be coagulated. This, as well as the supposition of Hippocrates, that such a degree of heat would dissipate the thin and watery parts of the blood, and in consequence gradually thicken the whole mass, was only an hypothesis, unsupported—and, indeed, disproved—by facts. Dr. Arnold, of Jamaica, remarks, that the assertion of Hales is irreconcilable with anything he has witnessed during a practice of twenty-five years. To what a degree that temperature reaches in extra-tropical regions, I am unable positively to state, not possessing facts of sufficient accuracy to enable me to form a correct opinion on the subject. Lining says it seldom exceeded 102 (p. 412), while Dr. Wragg writes that “among those who were hotter than is common, even in fever, the thermometer went up to 108° when placed in the armpit, and 105° on the cheek of a few of the hottest.”¹ In the West Indies, if we could credit Savarésy, we should conclude that the heat rises often as high as 38 or 40 degrees of Reaumur—equal to 117 or 122 Fahrenheit. But, admitting this to be an exaggerated statement, and relying on the more accurate experiments of Arnold, we shall find that, though the temperature falls short of the above, it is nevertheless very high, ranging in general from 104 to 110 (pp. 22, 28). While the heat is thus increased, the sensation imparted by the touch is often of a peculiarly disagreeable kind. We have already seen that it is by some called acrid; Rochoux (p. 365) calls it *âpre*; Archer, “an intense burning heat” (p. 66). Deveze speaks of the heat being “*violente, sèche, âcre, qui faisait éprouver une chaleur toute particulière au médecin*” (p. 22). Dr. Shecut remarks, that it leaves on the fingers, after taking the pulse, a sensation peculiar to this state of fever (p. 119). Dr. Kelly also remarks, that it communicates to the hand a peculiar repulsive sensation (p. 379). In like manner, Dr. R. Jackson speaks of the pungency felt when the part is closely embraced by the hand, being “unpleasant to sensation” (i. 68, 93); and Dr. Arnold informs us, in reference to the fever of Jamaica, that the feel of the skin at this period (stage of reaction) imparts a sensation so peculiar that words are wanted to convey a just idea of it to the reader (p. 22). “We feel, on the first touch, an involuntary desire to withdraw the hand, and a repugnance to renew the operation.” Pym also says, that

Sketch, pp. 46, 51; Moseley, p. 436; Hillary, p. 147; McArthur, p. 346; Savarésy, pp. 270, 1; Pugnet, pp. 353, 4; Blane on Seamen, p. 412; Gillespie, p. 38; Bancroft, p. 30; Comrie, xiii. 175; Frost, xiii. 29; Osgood, p. 9; Ralph, ii. 65, 67–8; Imray, liii. 79; Rufz, pp. 11, 13; Peixotto, i. 412; Barrington, xii. 311; Lefort (De la Saignée), p. 577; Pym, pp. 60, 228, 229, 233; Dariste, p. 159; Wilson, pp. 8, 20; Dyott, p. 1003; Belcher, p. 249; Gilbert, p. 65; Bally, p. 210; Caillot, p. 16; Morgan, iv. 4; Maher, p. 842; Dancer, p. 82; Leblond, p. 102; Desportes, i. 194; Rochoux, p. 365; Lefou-lon, pp. 68, 370; Fev. on board of Macedonia, p. 29.

¹ Charleston Med. J., x. 73.

the heat is intense and peculiar, and seems to adhere to the hand after having touched the body of the patient (p. 60). Such things must be familiar to those who have seen much of yellow fever—but frequent as they are, they do not appear to be essential to the disease, for Dr. Townsend informs us that in New York, in 1822, “the fever was rarely or never characterized by the biting, stinging heat of typhus” (p. 146); and similar observations have been made here and elsewhere.

Common, however, as cases may be, which are marked by increased temperature of skin, we frequently find the surface exhibiting a different character; and as the pulse differs in point of strength and volume in the first stage of the yellow fever, so also the temperature of the skin varies considerably under like circumstances; for, while in many cases it is considerably elevated, pungent, and burning, in a larger number the heat is moderate, or exceeding but little the standard of health. Again, in other instances, when the disease is characterized by broken or imperfect reaction, and especially in those of a decidedly congestive nature, the skin is cool, or even preternaturally cold from the onset of the attack. Dr. Rush noted such a condition of skin in the epidemics of 1793 and 1797 (iii. 68, iv. 12). It is mentioned also by Deveze (p. 29) and Cathrall (p. 23). Dr. Caldwell states that, in 1805, cases occurred in which the skin remained of natural temperature (p. 86). Similar facts are mentioned by Dr. Jackson (p. 52), and were observed in 1853–4. Cases which have occurred elsewhere in this country have been dwelt upon by various authors.¹

On this point, as on all others, the fever of the United States approximates to that of other countries; for, in Spain, Dr. R. Jackson found that in some forms of the disease the heat was rarely above the natural standard—sometimes below (p. 81). The epidemic of Barcelona, in 1821, was mostly characterized by a like condition of the temperature;² while the former epidemics of Spain, and those of Gibraltar, have furnished very many examples of the same sort.³ Nor is it less true that we may easily adduce proofs, from the writings of those who describe the yellow fever of intertropical regions, that, in respect to natural, or even low temperature of the skin, from the outset to the close of the attack, the disease is in no way different from our own or that of Europe. Dr. R. Jackson has, on this subject, furnished us the most convincing proofs of the correctness of the statement; showing, as he does, that, at the invasion of one of the varieties into which he divides the disease—that manifesting its action in the gangrenous temperament—the superficial heat is rarely increased beyond the natural standard; sometimes it does not

¹ Lining, ii. 418; Dickson, p. 254; *Ibid.*, *Eclect. J.*, iv. 110; Tully, p. 297; Girardin, p. 56; Hosack, p. 388; Barton, p. 15; Pascalis, *Mars. J.*, v. 144; Merrill, ii. 223, ix. 245; Cartwright, p. 13; Revere, iii. 224–5; Archer, v. 68; Bailey, p. 96; Stone, ii. 558; Harrison, p. 133, &c.

² Rochoux, p. 470; Audouard, pp. 56–58; Pariset, p. 394.

³ Caisergues, pp. 168–170; Fellowes, p. 54; Gillkrest, pp. 270–273; Velasquez, *Pariset*, p. 10; *Pariset, Observ.*, p. 30; Smith, *Edinb. Journ.*, xxxv. 42; Louis, p. 169; O'Halloran, p. 79.

attain that standard on the extremities, while it is high, pungent, and acrid on the trunk and about the præcordia.¹ In the concentrated form of the same variety, the external heat is seldom high, as judged by the hand, or as measured by the thermometer applied to the exterior (i. 90). During the period of excitement, "the skin is generally thick and torpid, not warm" (i. 92). In another variety—that manifesting itself in the phlegmatic temperament—the heat is rarely high, sometimes less high than natural on the extremities (i. 104). Again, in the concentrated form, "the heat which succeeds to the cold is slowly established; when established, it is seldom of a high scale on exterior surfaces" (i. 108). Than this, nothing can be more satisfactory, corroborated, as it is, by other authorities. Lempriere states that in his hybrid form—that which corresponds to the malignant form of other writers—"the heat is not intense" (ii. 83). Dr. Steward speaks of the cool skin as prevalent in the fever of Grenada in 1793–1794.² Bally tells us that the heat was seldom great in the fever of St. Domingo (pp. 19, 210, 267). Frost informs us that, at Demarara, the heat of the skin was often "gentle, not much exceeding that of nature."³ Dr. J. Clark found it moderate—not so great as a hot fit of an intermittent, or not much above the natural standard (pp. 7–9). Madrid, speaking of the yellow fever of Havana, says: "Color, aunque no halituoso como en las fiebres inflamatorias" (p. 24); and in another form he found "el color no es tan sensible, ni tan constante, antes es muy irregular" (p. 30). Osgood, in reference to the fever of the same place, says that sometimes there is little outward, but great inward heat (p. 14). Ralph tells us that in some cases, during the epidemic of Barbadoes in 1816, the temperature was moderate. Imray observed, in Dominica, that the surface in bad cases was constantly, but not much, above the natural standard (liii. 79). Wallace remarks of that form of the disease which he admits to be the effect of miasm, that "the heat of the surface may be higher than natural, but it is not the burning heat produced by intense action" (xlvi. 275). Lefoulon states that the skin is not hot (p. 68). Blair says it is sometimes damp and cold (p. 64). In many cases observed at Sierra Leone in 1823–1829, the temperature was considerably below par. (*Boyle*, p. 290.) And Dr. Wilson states that, in the congestive form, the heat of the surface is frequently less than in health. When it equals or surpasses the healthy standard, as it sometimes does—being highest about the epigastrium—it is of a peculiar kind. The hand is scarcely impressed by it, when applied lightly and hastily to the pit of the stomach; but, when kept there with steady pressure, a sensation of deep-seated and accumulated heat is communicated (p. 20). The temperature is deceptive. In some varieties of the fever described by Jackson, "the heat of the surface and extremities, especially as lightly touched, is moderate; as closely embraced, pungent and unpleasant to sensation by its impression" (p. 51). Dr. Barry, in his report on the epidemic yellow fever of Sierra Leone in 1823, already mentioned, says: "There was no degree of heat or dryness of the surface

¹ Sketch, &c., i. 87.² Med. and Philos. Register, iii. 184.³ Repos., xiii. 33.

of the body; but, on grasping the limbs or body firmly, a very peculiar sensation of stinging heat was communicated to the hand, which it retained for a considerable time." (*Boyle on African Fever*, p. 271.)

It may be remarked that the temperature of the skin appears, in many cases, to have no connection with the state of the arterial system. Chisholm informs us that he found the skin to be warm while the pulse was at 52, and that it was sometimes disagreeably cold when the pulse was as quick as in ordinary fever. Dr. Rush made a similar remark in 1794, and mentions a case in which the heat over the whole body was intense at a time when the patient was pulseless (iii. 212). He also speaks of cases marked by *cold sweats*, though the pulse was strong (iii. 63). And in 1820, it was not uncommon to find cases attended with entire torpor of the cuticular capillaries and coldness of the surface, even when the heart and large arteries were acting violently. Like phenomena were noticed in 1853 and 1854. By other writers, on the contrary, the connection between the temperature of the body and the force of the circulation is considered as well established from the facts they have observed. Such is said, by Dr. Townsend, to have been the case in New York in 1822.¹ To this conclusion we might come from the result of the experiments of Arnold, who found that the temperature was highest in individuals in whom the pulse beat frequently and actively. My own observations lead me to differ from Dr. Townsend on this subject. According to Dr. Blair, the heat is greater when the head is affected, or when there exists an inflammatory complication (p. 77).

B. *Condition of the Cutaneous Secretions*.—The condition of the external surface of the body does not vary, during the early stage of the yellow fever, in regard merely to the degree of its temperature. It does so, to an equal extent, relative to the condition of its secretory functions; for while, in many cases, during all seasons and everywhere, and at other periods almost universally, the skin at the outset, or throughout, is found dry, in other instances it presents, under similar circumstances, a greater difference of character. The dry skin, combined generally, though not necessarily, with increased heat, was observed in our early and all subsequent epidemics, and forms a prominent feature in the description of them contained in the writings of Rush. This eminent physician says that, in 1793, "in general, the skin was dry in the beginning, as well as in the subsequent stages of the disease" (iii. 63). In 1794, the fever often wore away without the least moisture on the skin (iii. 208). In 1797, "there was seldom any moisture, or even softness of the skin" (iv. 11). Currie (*Fev. of 1793*, pp. 21–30), Deveze (p. 22), Nassy (p. 22), take no notice of any other condition of the skin; while Jackson (pp. 52, 59, 61) describes it as of frequent occurrence. The same condition of the surface prevails also in the fever of other parts of this country.²

¹ Townsend, p. 146.

² Dickson, *Med. and Phys. Journ.*, iii. 254; Lining, p. 412; Moultrie, p. 3; Shecut, p. 119; Hayne, *Charleston Journ.*, vii. 4; N. O. in 1819, p. 8; Harrison, p. 132; Bayley, p. 96; A. Hosack, p. 11; Davidge, p. 102; Brown, p. 12; Monson, p. 179; Stone, pp. 181, 554.

The physicians of Leghorn,¹ at the same time that they found the skin to exhibit an elevated temperature, found it also often dry. The same condition is noticed in the accounts we have of the fever of Spain.²

In tropical regions, also, the dry—even parched—skin at the outset, or throughout, is not an unfrequent attendant on the disease, and has been, and continues to be, noticed by most writers.³

But the dry skin, though common in the yellow fever, is not universally found to characterize the first stage of the disease. In many instances, the surface, whether hot or otherwise, imparts an unctuous, greasy feel to the touch; or is moist—partially or all over. It exhibits even a disposition to free perspiration, and in some cases is bedewed with sweat from the accession of the attack, or after a short period of dryness—a condition which often remains unchanged throughout, or, as sometimes happens, is lost as the disease progresses. Such instances are observed during all sickly seasons in this country and elsewhere. They have been occasionally noticed in this city in 1793–'4,⁴ in 1805,⁵ in 1820,⁶ and in 1853, as well as in other American cities, from the days of Lining to the present.⁷ During the epidemic of Barcelona in 1821, the skin was generally moist from the onset, or even covered with perspiration.⁸ And if we pass from temperate to tropical regions, we shall discover that there also the disease is not unfrequently accompanied with a moist or perspirable skin—partially distributed, or universally diffused over the body; limited to a short period, or continuing throughout the course of the attack; and sometimes conjoined with increased, at others with a natural or decreased degree of temperature. “The skin,” says Warren (p. 9), “is sometimes, though rarely, parched and dry; but oftener, and indeed generally, moist and disposed to sweat.”⁹

As we have seen, these opposite conditions of the skin are intermixed in equal proportion, or nearly so, during some sickly seasons. At other periods, the one preponderates over the other, most of the cases being characterized either by a dry or by a moist surface; and, while the fever in certain places may more frequently than in others, assume one of those peculiarities, the differences in question are sometimes noted at various points during the same season, or in the same place during different seasons. In 1820, the fever, both in this city and at Middletown, Connecticut, exhibited the pecu-

¹ Tommasini, pp. 9, 10, 137; Dufour, iv. 51.

² Doughty, pp. 126–149; Caisergues, p. 167; Berthe, p. 84; O'Halloran, p. 126.

³ Dariste, p. 159; Dyott, p. 1003; Gilbert, p. 65; Belcher, p. 249; Rochoux, p. 321; Morgan, *Med. and Phys. J.*, iv. 4; Maher, p. 848; Rufz, p. 11; Bancroft, p. 9; Moseley, p. 436; Dickinson, p. 125; Fev. on board of the *Macedonia*, p. 29; Joubert, p. 966; Finlay, p. 12.

⁴ Rush, iii. 63, 208.

⁵ Caldwell, p. 81.

⁶ Jackson, pp. 52–59.

⁷ Lining, p. 412; Moultrie, p. 3; Bayley, p. 96; Dickson, *Eclect. Journ.*, iv. 409, 410; N. O. in 1839, p. 321; Tully, p. 297; Stone, 181, 554; Harrison, p. 131; Wragg, p. 73.

⁸ Rochoux, p. 470; Pariset, p. 394.

⁹ Dariste, p. 159; Gilbert, p. 65; Caillot, p. 17; Rochoux, p. 322; Rob. Jackson, p. 105; Maher, p. 848; Rufz, p. 11; Osgood, p. 9; Bruce, p. 278; Joubert, p. 967.

liarity in question. In the latter place, "the skin was uniformly either of a silky or of a leathery feel."¹ In this city, the malignant and mild cases were often marked by dryness; but often also there was considerable moisture of the surface. If not positively moist, it easily became so under the hand. In some cases, free clammy sweat continued nearly the whole time.² According to Dr. Lining, the skin, during the epidemic of 1748 at Charleston, was generally moist, and disposed to sweat (p. 412). In the epidemic of the same city in 1817, Dr. Dickson found the surface mostly hot and dry;³ while, in speaking of the fever of 1838, the same physician remarks, that it was a matter of common remark with the physicians whom he most commonly met during that season, that the skin of the yellow fever patients was apt to be moist, and by no means especially hot—a circumstance which, on all former occasions since 1817 inclusive, had not been noticed.⁴

In some cases, whatever be the absence of the cutaneous secretions at the onset of the disease, a moisture or perspiration comes on after a shorter or longer period. It often proves salutary, especially if combined with other symptoms of a favourable character—and among these a *moderate* degree of heat—and puts an end to the attack. More generally it proves beneficial when it occurs at a subsequent period, though not often later than the end of the first stage. "Many people," says Dr. Rush, "were relieved by copious sweats on the first day of the disease. They were in some instances spontaneous, and in others they were excited by diluting drinks, or by strong purges" (iii. 63). They were more common in 1794, but on that occasion seldom terminated the disease (iii. 208). In 1797, on the contrary, such a disposition was not common, except in very mild cases (iv. 32–3). The same effect is referred to by Deveze (p. 46), Monges (ii. 58), Currie (pp. 23, 35–6; *Ib. on Bil. Fev.*, p. 219), H. McLean (p. 95). A like occurrence has often, though not uniformly, been observed in other places, in so marked a degree, indeed, as to cause free perspiration, or more frequently simply moisture of the surface, to be regarded when occurring at that time as critical, or at least as a favourable sign.⁵

But, whatever be the condition of the skin, both in regard to temperature and secretory action, during the first stage of the yellow fever, a considerable, and sometimes a rapid or sudden change takes place in these respects at the period of the metaptosis or remission which, as we have seen, most generally takes place on or about the third day. From hot and dry, the surface becomes of a natural temperature—soft, moist, and perspirable. If cool, or bedewed with sweat, it often becomes warm and drier. Such has been the

¹ Tully, p. 297.

² Jackson, pp. 52, 9.

³ Med. and Phys. J., iii. 254.

⁴ Eclectic J., iv. 109, 110.

⁵ Amiel, p. 167; H. McLean, 11, 95; Palloni, p. 6; Fev. of N. O. 1839, p. 338; Townsend, p. 155; Caillot, p. 158; Bally, pp. 473–4; Ticknor, iii. 226; Arejula, p. 181; *Ib.* Ed. J., i. 449; Caisergues, pp. 173, 180; Arnold, p. 17; Valentin, p. 173; Bancroft, p. 47; Berthe, p. 102; Audouard, pp. 59, 60; Pariset, Obs., pp. 14, 31; Gillkrest, p. 274; Blin, p. 11.

character of the change in the yellow fever of this city and country.¹ It has ever been found to be a characteristic of the yellow fever of the South of Spain. It was noted at Leghorn,² and has been dwelt upon, with few exceptions, by all writers on tropical fever.³

That few instances occur in which the change alluded to does not take place—the skin continuing unaltered, especially in point of temperature—all must admit. But these constitute exceptions to the rule. They occur in cases of a severe character, but are not unfrequently found also in mild cases when the febrile paroxysm is of longer duration, and with it the disease subsides completely in the way observed in other febrile affections. Be this as it may, the change is one of very usual occurrence. Taking place at the same time with the change in the pulse, noticed in the preceding chapter, it will not be necessary to point out at what period it usually occurs. From the time of the occurrence of this peculiar remission, the skin, excepting in a very few instances, never reacquires a higher temperature than natural. So far from doing this, it gradually loses the small degree of heat to which it had been reduced, until, at the approach or moment of dissolution, it becomes of an icy coldness; often, though not always, covered with clammy sweat.⁴

What we have seen in reference to the pulse we may see in reference to the skin, so far as its temperature and secretory functions are concerned. These are not necessarily implicated in the disease—remaining, as they do, in many cases attended with danger—even in those ending fatally, unaltered from their natural standard. The mischief produced by the impress of the efficient cause progresses in the vital organs, even to the extent of occasioning dissolution; and yet, while this is going on, and a concatenation of other symptoms portends the approach of death, the surface, like the arterial circulation, remains unchanged. In other instances, modifications in its functions manifest themselves; but, even in these, such modifications are often of a trifling character, and in no way commensurate with the import-

¹ Rush, iii. 73; Currie, p. 24; Cathrall, p. 27; Caldwell, p. 84; Jackson, p. 57; Drysdale, i. 29, 132; Irvine, p. 29; Lining, p. 418; Cartwright, p. 13; Townsend, p. 149; Girardin, pp. 34–5; Archer, p. 67; Smith of Galveston, xxv. 502; Warren, in Tytler, p. 502; Seaman, Repos., p. 249; Shecut, p. 119; N. O. 1820, p. 9.

² Rochoux, pp. 459, 470; Audouard, p. 58; Caisergues, p. 168; Berthe, p. 85—(the two last are not explicit, but mention diminution of the symptoms); Rayer, p. 21; Gillkrest, p. 270; Velasquez, p. 10; Fellowes, p. 53; Pariset, p. 400; R. Jackson, p. 74; Palloni, p. 4.

³ Moseley, p. 437; Bruce, p. 278; Fontana, p. 73; Jackson, Tr., p. 260; Sketches, pp. 70, 93; Blane, p. 412; Gillespie, p. 40; Bancroft, p. 11; Lempriere, ii. 61; Savarésy, p. 6; Ralph, p. 66; Imray, p. 80; McArthur, p. 347; Madrid, p. 6, 25; Chisholm, i. 150; Essay, p. 107; Hillary, pp. 149, 151; Osgood, p. 11; Barrington, xii. 312; Wilson, p. 10; Frost, xiii. 29, 30; J. Clark, pp. 10, 15; Dariste, p. 161; Bally, p. 210; Dyott, p. 1003; Belcher, p. 250.

⁴ Moseley, p. 438; Chisholm, i. 149; Pugnet, p. 351; R. Jackson, i. 93, 111; Vatable, p. 346; Currie, pp. 25–6, 220; Cathrall, p. 31; Jackson, p. 52; Cartwright, ix. 10; Revere, iii. 225; Townsend, p. 159; Drysdale, i. 29, 132; Dickson, iii. 255; Pariset, p. 417.

ance and severity of the attack. They show the absence of harmony between the implication of the skin and that of other parts with which the former, in other complaints, usually sympathizes in a marked degree. In some instances, it is true, the skin is over-excited or greatly depressed; it is much hotter or much colder than in health; it is parched and dry, or bedewed with perspiration. But such phenomena are often wanting, and are as frequently—indeed, more commonly—encountered in other complaints of a different kind. When they occur in the yellow fever, they may, in a certain measure, be regarded as exceptional. They serve in no way to distinguish or characterize the disease; and may, perhaps, without fear of error, be regarded in the light of complications, the results of inflammatory or nervous irritation in some instances, and of nervous atony in others; consequent, occasionally, on the reaction succeeding to the depressing influence of the remote cause, or to the speedy and deadly effect of the latter. But be the process of their production what it may, such derangements in the skin are not necessary attendants on the disease, which often—indeed, generally—proceeds from the outset to the close of the attack without exhibiting any considerable, if any change in the temperature or secretory functions of the covering membrane.

c. *Odour of the Skin.*—Many writers on the yellow fever of temperate and tropical regions have taken no notice of the odour emitted by the skin during the progress—though particularly during the latter stages—of the disease; and there are not wanting those who have expressed a doubt as to the reality of anything peculiar in that respect, or simply denied its occurrence in the cases that presented themselves to their observation. Dr. Townsend, for example, in his account of the epidemic of New York, in 1822, says that there was not, at any time, as far as his experience went, a foul cadaverous effluvium from the body, “a symptom so peculiarly characteristic of typhus, with which yellow fever, however, ought not to be confounded. On the contrary, in a majority of cases, everything about the patient seemed perfectly inodorous, and for hours after death.”¹ Dr. Tully, also, in speaking of the fever of Middletown, Conn., in 1820, informs us that, in general, there was but little fœtor before death, and even the usual febrile smell was not, in many instances, perceptible.²

But whatever may have been the case in New York and Middletown, at the periods in question, and in other instances that might, if necessary, be mentioned, and however true it may be that from these facts and the silence of several writers on the subject, we might infer that the surface of the body in the yellow fever does not emit an odour of a peculiar kind; yet facts sufficient may be gathered from the writings on this disease, in both temperate and tropical regions, to bear me out in what is here stated, and to show, not only that the symptom in question occurs often, but does so under circumstances calculated to induce us to view it as pathognomonic of the fever

¹ Op. cit., p. 165.

² Essays on Fevers and other Medical Subjects, &c., p. 30.

generally. I cannot doubt having recognized a peculiar and offensive odour about the persons of individuals affected with this disease; and I think it has aided me, in some instances, in establishing a correct diagnosis. In one or two cases, the smell was highly offensive. It has been noticed in all our epidemics. Dr. Deveze¹ speaks of it as of frequent occurrence in the latter stages of the disease, and designates it as fetid—*une odeur infecte*. Dr. Rush² states that, in 1793, the sweats in some persons had an offensive smell resembling that of the washings of a gun. This odour was emitted by those who, though not ill of the fever, were exposed to the prevailing cause. It was perceived even in those cases in which the disease went off on the first day of the attack.³ In his history of the epidemic of 1794, the same eminent physician says: “I recollect having more than once perceived a smell which had been familiar to me during the prevalence of the yellow fever in 1793. It resembled the smell of liver of sulphur. I suspected for awhile that it arose from the exhalations of the gutters of the city. But an accident taught me that it was produced by the perspiration of my body. Upon rubbing my hands, this odour was increased so as to become not only more sensible to myself, but in the most sensible degree to my pupil, Mr. Otto. From this fact, I was convinced that I was strongly impregnated with miasmata.”⁴ Dr. S. Jackson mentions that, in one case, a most intolerable fetor proceeded from the patient’s body for twenty-four hours before his entire and complete dissolution.⁵ Drysdale,⁶ in Baltimore; Hill,⁷ in Wilmington, North Carolina; Gros,⁸ in New Orleans; Stone,⁹ in Woodville and Natchez; A. Hosack,¹⁰ in New York; Monson,¹¹ in New Haven; Dickson,¹² in Charleston, have made similar observations during the epidemics they describe. During the disastrous epidemic which desolated a large portion of the southern section of this country in 1853, and extended far and wide, the peculiar odour under consideration was noticed, and is mentioned not only by Dr. Barton, in his able Report on the Sanitary Condition of New Orleans, but by a large number of the physicians from whom information was solicited—Drs. White, Jones, Brown, Richardson, Grant, and Benedict. It was also noticed by Dr. W. Humboldt, in Mexico.¹³

Speaking of the fever of Woodville (1844), Dr. Stone remarks: “Sometimes before, and always after an attack, and not dependent upon the perspiration, a peculiar odour was perceptible, which it is not easy to describe; but which to have observed once is to remember always; I think I could detect the disease by this alone. One of my children, aged eight years, gave off this odour twelve days before the development of fever.” Besides this, the perspiration was of an offensive odour. Of the symptom under present

¹ *Traité de la F. J.*, p. 26.

³ *Ibid.*, iii. 63.

⁶ *Fever of 1820*, p. 54.

⁷ *Med. Recorder*, v. 90.

⁹ *N. O. J.*, ii. 180; *Ibid.*, v. 453.

¹¹ *Webster’s Collection*, p. 182.

¹³ See Report, pp. 13, 29, 34, 35, 45, 82, 127, 245.

² *Op. cit.*, iii. 85.

⁴ *Ibid.*, iii. 217.

⁶ *Med. Mus.*, i. 137.

⁸ *Rep. on Fever of 1817, N. O.*, pp. 12, 17.

¹⁰ *Y. F. of New York in 1795*, p. 16.

¹² *Essays*, p. 373.

consideration, as it presented itself four years after in Natchez, the same writer says: "An odour which I cannot describe, but which was *the* odour that had been indelibly impressed on me in 1844, was strongly perceptible in many of the simplest cases towards the latter part of the epidemic, but which I did not notice for a month after its commencement." On this, as on the former occasion, the "perspiration was always offensive, often fetid."

In the yellow fever epidemics of Leghorn, and of various parts of the South of Spain, the same symptom was frequently observed, and is specially noticed in the numerous accounts of them which we possess. In the first-named city, the fœtor was compared to that of fetid bile.¹ Arejula,² at Malaga, in 1803, likened it to the smell of putrid fish. It is mentioned by the same writer, Berthe,³ and others, as of common occurrence during the epidemics of Andalusia, from 1800 and 1810. Martel,⁴ in describing the disease as it occurred among the French soldiers in 1811, at Rotana, Levrilla, and Alcantarilla, lays great stress on that symptom. Dr. R. Jackson⁵ informs us that, at Cadiz and Xeres, in 1820, the fœtor emitted by the body of the sick, in some forms of the disease, was singular—sickly and faint, and not unlike the smell of a fish-market. The next year, at Barcelona, Pariset⁶ and his colleagues noticed a cadaverous odour in some, though not generally. Andouard⁷ mentions it as of frequent occurrence; so does Lafuente,⁸ and other Spanish writers. Rochoux⁹ states that some of the sick emit a very offensive odour, analogous to that of gangrene; and adds that it is a usual attendant on the cutaneous exhalation, whether the latter assumes the character of sweat or of insensible perspiration. "It is not," he adds, "so strong as to be perceived from the street, as some physicians have asserted; but we never fail to be struck with it when we uncover a patient and approach very near him. It is of an insipid nature (*fade*), somewhat nauseous, and adheres strongly to clothes." The same writer remarks, in addition, that, on this point, the yellow fever approximates closely to hospital typhus, and the plague, both of which are characterized by a peculiar odour. In his account of the fever which occurred at Port de Passage in 1823, Dr. Jourdain remarks that the patients emitted a peculiarly disagreeable odour—insipid, nauseous, *sui generis*; that it occasioned gastric uneasiness, and that Dr. Arruti experienced from it nervous tremors.¹⁰

Nor is this all; Desportes,¹¹ more than a century ago, noticed the same phenomenon in the fever of St. Domingo, where it was subsequently observed by Bally¹² and Gilbert,¹³ the former designating it as fetid, the latter

¹ Edinb. Journ., ii. 84.

² Ibid., i. 448.

³ Op. cit., 55, 56.

⁴ Quoted by Bally, p. 250.

⁵ Epid. Y. F. of the South Coast of Spain, p. 105.

⁶ Op. cit., p. 435.

⁷ Op. cit., pp. 211, 393.

⁸ Observaciones sobre la Fiebre Amarilla, &c., Periodico de la Soc. Méd. Cir., i. 165. (See Rochoux, p. 472.)

⁹ Rochoux, op. cit., pp. 461, 472, 473.

¹⁰ Ann. de la Méd. Phys., v. 260.

¹¹ Maladies de St. Domingues, i. 221.

¹² Typhus d'Amérique, 247, 248.

¹³ Hist. Méd. de l'Armée Française à St. Domingue, 66.

as cadaverous. Dr. R. Jackson¹ found in the West Indies, as he did in the fever of Europe, cases in which the perspiration had a peculiar smell, resembling that of a fish-market. In the fever of Dominica, as we learn from Dr. Imray,³ the odour of the cutaneous exhalation was often extremely disagreeable as well to the patient himself as his attendants. Additional testimony on this subject might be gathered from numerous writers.³

When we take into consideration the close alliance existing between the odour of the blood and that of the skin in the state of health, we may presume that the peculiar symptoms to which attention has been called, arises from the odour imparted to the circulating fluid by the febrile poison. We may even go so far as to aver, that each variety of malarial fever presents something peculiar in that respect, which, while enabling the pathologist to distinguish it from pneumonia and other true phlegmasiæ, affords him the means of pointing out, to a certain extent, at least, the particular nature of the case under examination, or the class to which it belongs. Speaking of the second period of the Niger fever, Dr. McWilliams says: "The principal complaint at this period was from the odour of the perspiration, particularly in those cases that subsequently proved fatal. I was not sensible of this peculiarity in the smell of the perspiration in my own case, but I perceived it very distinctly in several others." In the bilious remittent fevers of Ceylon and the West Indies, a peculiar smell emitted by the sick is alluded to by Dr. Millingen, and other writers.⁴

D. *Sensibility of the Skin*.—In some cases, the sensibility of the cutaneous surface is morbidly deranged—increased or diminished—to an extent very uncommon, if not unparalleled in other febrile diseases. Dr. Rush informs us that, in 1793, there was in many persons a soreness to the touch. In some cases, this extended to every part of the body, and, to such a degree, indeed, as to render the application of common rum to the skin, and even the least motion of the limbs, painful (iii. 64–6). In one case, in 1794, the soreness to the sense of touch was so exquisite, about the crisis of the fever, that the pressure of a piece of fine muslin upon the skin gave great pain (iii. 210). Again, in 1798, the whole body was affected in many cases with this morbid sensibility or super-sensation, so that patients complained of pain upon being touched, when they were moved in their beds. It was general, in parts to which blisters had been applied. It continued through every stage of the disease, even to within two hours of death, and was sometimes combined with absence of pulse and coldness of the extremities (iv. 42). Drysdale noticed a similar phenomenon in Baltimore, remarking that "the

¹ Sketch, i. 64.

² Edinb. Med. and Surg. Journ., liii. 80.

³ Gillespie, p. 34; Savarésy, pp. 273, 4; Madrid, p. 25; Vatable, p. 346; Frost, Med. Repos., xiii. 33; Comrie, Ed. Journ., xiii. 177; Ralph, Ed. Med.-Chir. Tr., ii. 75; Arnold, p. 10; Bryson, p. 96; Fever of Cayenne, 1850, p. 175; Fontanelle Bellevue, cited by Magne, Ann. Mar. iv. 1844, p. 551; Mabit, pp. 10, 11; Dupont, pp. 19, 20; Bourdon, p. 12; Bally, p. 248; Vincent, p. 25; Jolivet, p. 11; Joubert, p. 968; Copland, iii. 141; Bertulus, pp. 49, 81; Campet, p. 78; Addoms, p. 10; Pennell, Fev. of Rio, p. 6.

⁴ Second Report of Lond. Board of Health, p. 364; J. Clark, Fev. of Dominica, p. 20.

nerves, in some cases, acquired such a morbid degree of sensibility as to render the whole surface of the body sore to the touch.”¹ Dr. Davidge remarked, in the same city in 1798, that the sensibility of the surface of the body was exceedingly morbid, and, on the least touch, communicated uneasiness; this preternatural excitability he met with in a surprising degree (p. 104). Bally, François, and Pariset made a like observation at Barcelona, in 1821 (pp. 384–404); and Dr. R. Jackson speaks of cases in the West Indies, in which the skin exhibited the extreme tenderness in question, as if it had been bruised (i. 87). Dr. Caldwell speaks of the sensibility or soreness of the blisters, so exquisite as to compel the patient to cry out as often as they were touched. “In many cases,” he remarks, “that terminated fatally, this extreme, and, as I may call it, preternatural soreness, particularly of the blisters on the wrists, constituted a very striking symptom. Recoveries from it in my practice were very rare.”²

In a different set of cases, the skin exhibits a contrary condition, being insensible and unirritable even to a degree unobserved, under parallel circumstances in other fevers, and comparable only to what we notice in Asiatic cholera. Dr. Rush refers to this phenomenon as occurring in the fever of this city (iii. 64), and states that the insensibility was sometimes so great that blisters made no impression upon the skin (p. 69). But, it is more particularly dwelt upon by Dr. S. Jackson, who, in his account of the epidemic of 1820, represents this morbid condition as attaining, at times, so great a degree of severity, as to be attributable to no less a cause than the death of the surface (p. 52). This torpor of the nerves, or capillaries of the skin, existed in most of the malignant cases which occurred at the season mentioned, whether the mental faculties were impaired or not—sometimes on the first and second days, generally about the third. “I have seen,” Dr. Jackson adds, “in several cases, the patient remaining from twenty to forty hours in a comatose state; the senses obliterated; the extremities and surface cold, and all its functions destroyed. I have had turpentine and cantharides poured, when boiling, on the abdomen, and rubbed on the extremities for hours, and yet the slightest redness was not produced, nor any more effect than if it had been applied to a statue. From every appearance, animal life was wholly extinguished. The heart, in such cases, would sometimes act with great force” (p. 54).

This insensibility and unirritability of the skin is, in like manner, mentioned by Dr. Robert Jackson as occurring in several of the varieties into which he divides the fever of the West Indies. Speaking of the aggravated degree of the disease as manifested in the sanguine temperament, Dr. J. says: “The skin, in the first period, is usually dry; thick, as if preternaturally compacted; torpid, and little sensible to stimulation, &c.; it is, in fact, such as characterizes agonies of suffering rather than energies of circulation” (i. 67). In the next stage, the skin is “of so little sensibility, that the strongest blisters sear, but do not vesicate (i. 69), and gives the idea of its having lost connection with the living system (i. 71), and more resembling, in some cases, a dead

¹ Med. Museum, i. 127.

² Fever of 1805, p. 11.

hide than the skin of a living man—without sensibility or power of reaction (i. 111). In another variety (that manifesting itself in the serous temperament), the skin is often thick, deficient in sensibility, scarcely vesicated by the application of the strongest blisters; so compact and solid, in many cases, that the hottest baths, or the strongest stimulating liniments, aided by the most careful frictions, are not sufficient to excite a temporary moisture" (i. 140–1). In the concentrated form of the gangrenous species, it is generally thick and torpid (i. 93), and in the eaehectic form it is smooth and polished, "as if under the discipline of cosmetics" (i. 126).

Louis has noted the want of tone in the skin, a condition which is considered as depending on a suspension or perversion of friction, the skin being sometimes dry, and at others moist and clammy; and, when grasped in the hand, feeling like the skin of one who has ceased to breathe. Others have noticed it,¹ and it has been observed here in our late epidemic.

E. *Colour of the Skin*.—The skin, in the generality of cases of yellow fever, is found to vary, in regard to coloration, according to the stage or period at which it is examined, as well as to the form or variety of the disease. That the line of demarcation between these is not always easily made out at the outset of the attack, no one will venture to deny; for, excepting in a few instances, the entire cutaneous surface is, at that period, remarkably pale, and in some cases has a shrivelled appearance.² But, as the disease progresses, the distinction becomes obvious. In the inflammatory form, and when reaction has set in, the skin loses the pallor mentioned, and soon becomes more or less red. Nevertheless, though the change is to some extent discernible all over, it is principally marked in the face, which becomes flushed—generally to a high, at times to a very remarkable degree; the difference depending, in great measure, on the more or less sanguine temperament of the individual, and the degree of virulence of the inflammatory reaction. In instances of the latter kind, the face often becomes animated, of a deep-red, crimson hue, highly suffused with blood, vultuous, and shining; at others, it assumes a dusky, violet, bluish colour. In cases marked by a less degree of reaction, or in which that reaction is broken or of doubtful kind, the redness is of a more subdued character—sometimes of a rosy tint, rather pleasant to the sight than otherwise. Nor is the redness of the face completely absent in all cases marked by imperfect reaction; for in these it often presents itself in a higher degree than is observed in corresponding grades of other fevers. So common, indeed, is the unusual redness of the skin (of the face particularly), and so peculiar and marked is the degree it attains, that this symptom has commonly, and very properly, been viewed as pathognomonic of the yellow fever, and may alone, in many cases, indicate to an experienced eye the real nature of the disease; or, if not alone sufficient for that purpose, will ever be regarded in the light of a most important diagnostic sign.

The increased redness of the skin here alluded to—from a moderate red to a dark crimson tint—has been the subject of observation in every place where the yellow fever has prevailed, varying, however, in regard to degree, in

¹ Haynes, *Charleston Journ.*, vii. 3.

² Barnwell, p. 369; Nassy, p. 23.

various localities, or in the same localities at various periods. In this city, it has been described as of almost universal occurrence, and at times as manifesting itself in a marked degree. Dr. Rush speaks of the face being suffused with blood (iii. 52). In another place, he states that the redness was so intense (in 1794) as to produce an appearance of inflammation (iii. 211). Devezé, in like manner, found the face red, and, as it were, inflamed—*rouge et enflammée* (p. 22). Currie speaks of it as “highly flushed” (p. 21); and expressions of kindred, if not identical nature, may be found in the writings of Nassy (p. 21), Cathrall (p. 24), Jackson (pp. 51, 59, 61), and Caldwell (p. 85).

Equally marked has it been found in other parts of this country, from Boston to Galveston.¹ In the fever of Europe, it has been very generally taken notice of. In his account of the epidemic of Seville, in 1819, Dr. Velasquez, as quoted by Pariset,² remarks that in the commencement of the disease “the face and lips became inflamed.” Not different was it at Cadiz during the same season (p. 29). Speaking of the epidemic of that city, in 1800, Caisergues states that, as the disease progressed, the face became more animated and red (p. 167); and Berthe speaks of the intense redness of the face on the same occasion (p. 81). Arejula observed the same phenomenon at Malaga in 1803;³ as did also O’Halloran (p. 77) and Jackson (p. 105) at Cadiz and Xeres in 1820; Audouard (pp. 56, 57), Rochoux (p. 469), Pariset, Bally, and François, at Barcelona in 1821 (p. 384); Dufour (*Marseilles Journ.*, iv. 50) and Palloni (p. 6), at Leghorn in 1804; Louis (pp. 167–8, 172–5) at Gibraltar; and, if we inquire of tropical writers, we shall be told that the phenomenon, even to an intense degree, is of general occurrence in the cases that have fallen under their observation.⁴

But while the suffused skin, particularly that of the face, is acknowledged to be a frequent, indeed almost universal symptom of the first stage of the yellow fever, varying from a light rosy hue to a dark crimson or purplish or violet colour, other instances, particularly those of a malignant form, or when open reaction does not take place, and the patient passes at once from the prodromic to the last stage, the skin, in whatever part examined, presents a very different aspect. In these it is generally found but slightly, if at all coloured; sometimes of the natural hue, or even more or less pale. Cases of this kind are found wherever such typhoid and congestive cases of the disease

¹ E. H. Smith, p. 115; Drysdale, i. 29; Areher, v. 66; Hill, v. 89; Dickson, p. 254; Dalmas, p. 6; Gros, p. 9; Girardin, p. 33; Thomas, p. 82; N. O. in 1819, p. 8; do. in 1820, p. 8; Perlee, iii. 11; Ticknor, iii. 224; A. Smith, xxv. 501; A. Hosack, p. 11; Monson, p. 179; Moultrie, p. 3; Stone, ii. 180; *Ib.*, vi. 557; Wragg, x. 72.

² *Observ.*, p. 10.

³ *Edinb. Journ.*, i. 448.

⁴ R. Jackson, *Sketch*, ii. 62; Bruce, p. 278; Fontana, p. 72; Pugnet, p. 354; Bancroft, p. 30; Moseley, p. 436; Hillary, p. 147; Desportes, i. 194; McArthur, p. 346; Chisholm, i. 149; Savarésy, p. 269; Imray, liii. 79; Gillespie, p. 38; Osgood, p. 10; Ralph, ii. 70; Frost, xiii. 29; Hunter, p. 65; Rufz, p. 54; Vatable, p. 345; Caillot, p. 16; Davidson, viii. 248; Wilson, p. 8; Dancer, p. 82; Maher, pp. 842–848; J. Clark, pp. 6–8; Dariste, pp. 158, 159; Bally, p. 214; Catel, p. 11; Gilbert, p. 65; Dickson, in Johnson, p. 365; Rochoux, pp. 294, 295, *ib.* *Treatise*, p. 112; Pym, pp. 228–233.

present themselves. They were observed in Barcelona in 1821, as we learn from Dr. Rochoux (p. 468), and in Cadiz in 1820, according to R. Jackson (p. 62). Dr. Rush found the skin sometimes of a dusky colour (iii. 52). Currie speaks of the livid countenance (p. 28); Caldwell does the same (p. 83). Stone describes the face as being sometimes pale (p. 180). Dr. Merrill speaks of "the smooth, white, marble-like aspect of the skin."¹ Dr. Wilson calls attention to the fact that, in the congestive form of the West Indian fever, "the skin is of a decidedly pale or livid colour" (p. 11). Bally speaks of the cadaverous pallor presented in some cases (p. 314). Madrid informs us that, in some instances, there is a "color palido desde il principio" (p. 6). And Dr. R. Jackson tells us that, in some, the countenance is livid, as in sea-scurvy (i. 86); in others, "livid, and of a peculiar gloss" (i. 91); that, in a different set, the countenance is generally pale and inanimate (i. 96, 105); and that, in a different set, again, it is sometimes sallow and lurid (i. 109). Leblond states that the skin is sometimes pale and lurid (p. 104).

Be this as it may, a reference to all writers on the subject will show that the redness of the face or skin generally, when it does occur, continues during the whole of the stage of excitement, and beginning to subside at the accession of the metaptosis or remission, soon disappears, never, or seldom, to return, and gives way to a natural colour of the parts or to other phenomena of a different kind, which will be taken notice of in the succeeding chapters.

CHAPTER IX.

STATE OF THE SKIN—CONTINUED.

JAUNDICE.—The redness of the first stage having subsided at the period mentioned; or, in cases attended with paleness of the skin, the disease having progressed for a longer or shorter space of time, we observe that peculiar discoloration of the surface from which the disease derives the name by which it is at present almost universally designated.

Natural, however, as it might be to infer, from the very general adoption of the name of yellow fever, that the symptom in question—jaundice—is a constant attendant on the disease, experience has amply shown that such is far from being the fact, and that, in a large number of instances, the case proceeds to a favourable or fatal issue, without being characterized by the phenomenon in question. This, as will be perceived, completely disproves the assertion of those who, like Fournier Pescay,² have, in utter ignorance or disregard of innumerable and incontrovertible facts, collected in every spot where the yellow fever has been known to prevail, ventured to maintain that the morbid process which constitutes the fever under consideration,

¹ N. Am. Med. and Surg. Journ., ii. 223.

² Dict. des Sci. Méd., xv. 340.

never fails to impress a yellow colour upon the skin, and thereby justifies the name assigned to it. Nothing, however, as has long since and repeatedly been pointed out in this country and elsewhere,¹ is more evident than the impropriety of the name of yellow fever; inasmuch as the peculiar discoloration, from which that name is derived, is not pathognomonic of the disease; for, not only is it, as already said, absent in very many cases throughout the whole course of the attack, but, as we shall see, it often does not show itself until after the death of the patient, or at a period when the true character of the disease has already been fully made out; while, again, yellowness of the skin is an attendant on other complaints having little, if any, pathological and etiological connection with the yellow fever. In a word, if the Spanish physicians in Europe and South America see many cases of *vomito negro* without black vomit, so English, French, and American physicians frequently notice cases of yellow fever without yellow skin; and everywhere diseases that are not yellow fever are attended with yellowness of the eyes or skin, or both.

That jaundice, even when of the most marked kind, is not by itself sufficient to enable us to identify a case of yellow fever—in other words, that it is not perfectly characteristic or pathognomonic of the disease, is, I repeat, evident from the fact that it exists in many febrile and other complaints which have little or no pathological and etiological connection with it. We need not stop to show that it is a common or universal symptom in many of the modifications of hepatic disease—acute and chronic; and in mechanical obstructions to the passage of bile into the intestines. In some cases of plague, a yellow suffusion, similar to that so often observed in the yellow fever, occurs. It may be, and doubtless is, rare in that disease; but its existence, even in a few cases only, indicates that it is not characteristic of the yellow fever. The icteric hue of the skin in like manner shows itself occasionally in Asiatic, as well indeed as in common cholera. The *causos* of Hippocrates, or inflammatory fever of other authors, is—especially that form of it observed in tropical regions—characterized by the symptom in question. From the accounts we have of the symptomatology of the relapsing fever of Scotland and Ireland, we gather that in a certain number of cases, on the third or fourth day, the conjunctiva, as well as the skin—particularly of the face—assumes a slight bronze tint. This is followed, on the fifth or sixth day of the disease, by an attack of jaundice, the skin being often of a brassy, turmeric, yellow colour, attended with severe vomiting, &c. It is most intense on the face; next, on the abdomen and thighs. The hue of the neck and chest is the most vivid; then comes, of equal, or nearly equal brightness, the abdomen; then, somewhat fainter, the thighs; then, considerably paler still, the legs, arms, and forearms. The hands and feet get their colour later, always to a much less extent, and sometimes not at all.²

¹ Deveze, p. 4; Barnwell, p. 372; Drysdale, i. 132; R. Jackson, *Fev. of Spain*, p. 52; Pinckard, *Notes on West Indies*, ii. 227–263, 1st edit.

² Cormack, *Nat. Hist. Pathol. and Treat. of the Epid. Fever of Edinb.*, &c. p. 23; Jenner, *Med. Chir. Trans.*, xxxiii. 26; Steel, *Edinb. Med. and Surg. J.*, lxx. 165; Henderson, *ib.*, lxi. 220, 225; Craigie, *ib.*, lx. 412; David Smith, lxi. 69, lxii. 64–5; *Brit. and*

This is certainly not very different from what happens in yellow fever, and yet, as I shall have occasion to show in a future chapter, the latter cannot be confounded with the relapsing fever, from which it differs essentially on many points. In some cases of the typhus of England and Ireland, the general surface of the body exhibits—particularly after death—a dirty purple yellow hue,¹ with no other phenomenon calculated to raise the least suspicion of any close analogy between this fever and the one which forms the subject of our present inquiries.

The fever which prevailed in this city among the blacks in the summer of 1820, and which, in consequence of the race of individuals it selected, received the name of the negro fever, as well as that which spread in some parts of New York in 1822, and was called, from its main location, the Bancker Street fever, were modifications of malarial remittents, and but remotely connected with the yellow fever. In both, the icteric hue was common—recognizable in the whites, some few of whom were attacked here by the former disease, and many in New York by the other—over the face, trunk, and eyes, and in the blacks in the latter organ only.² In the Chagres remittent, jaundice plays a conspicuous part, and constitutes one of the main points in the arguments of those who contend for the identity of this and the true yellow fever.³ So, also, in the African remittent, in which yellowness of the skin and conjunctiva is of at least occasional occurrence.⁴ In the endemic Pucca fever of Bengal, “a yellowish suffusion of the skin *as in the endemic of the West*,” is not unfrequently observed. Dr. Johnson, who makes this statement, adds: “This suffusion of bile, or yellow colour on the skin, is by no means an uncommon symptom in the fevers of the East.” “The natives themselves frequently exhibit this appearance when extensive epidemics prevail in the lower situations of Bengal.” Capt. Williamson says, in relation to this subject: “Certainly it is common to see whole villages in a state of *jaundice*, and in some years the ravages of the disease (marsh fever) are truly formidable.”⁵ It was noticed in the epidemic of Coimbatore (p. 97), Seringapatam.⁶ In the Edam fever described by Johnson, “a great proportion changed, in a few days, to a bright yellow; some to a leaden colour” (p. 130). We all know that jaundice is a common symptom in the remittent of Batavia,

For. Med.-Chir. Rev., viii. 7; Alison, Scottish and North of England Med. Gaz., i. 2; Reid, Lond. Med. Gaz., xxxiii. 359.

¹ Reid, Tr. of College of Phys. of Ireland, v. 271; Huxham, ii. 205, 252; Armstrong on Typhus, p. 59; O'Brien, Trans. of College of Phys. of Ireland, v. 531-2; Barker, ib., iii. 563; Stoker, ib., iii. 455; Cheyne, Dublin Hosp. Repts., ii. 20, 68; Barker and Cheyne, An Account of the Rise and Progress of the Fever lately prevalent in Ireland, i. 251, 434; Shannon, Pract. Obs., xii. 27, 167; Southwood Smith on Fever, p. 54.

² Klapp, Med. Rec., iv. 85; Emerson, Phil. Med. and Phys., iii. 201.

³ Finlay on Rem. and Y. Fevers, p. 18.

⁴ Pritchett, Some Acct. of the African Rem. Fev., p. 156; McWilliams, Med. Hist. of the Exped. to the Niger, p. —; Boyle, Acct. of Western Coast of Africa, p. 129; Lind, pp. 61, 71; Bryson, pp. 66, 70; Robertson in Lind, p. 66.

⁵ Johnson on Trop. Cl., p. 47; Shannon, op. cit., pp. 12, 27, 167, 171.

⁶ Nicoll, Edinb. J., xi. 280; Johnson, p. 100; Trans. of Med. and Phys. Soc. of Bombay, No. 6 (1843), p. 189.

and in the jungle Wynadd fever of Bombay, Madras, and Malabar, and of the Coromandel coast. It was a prominent symptom in the Rohilcund fever.¹ The remittent of Ceylon is in like manner marked by yellow colour of the skin.² In his interesting report on the epidemic ague, or fainting fever of Persia, which occurred in Teheran in the autumn of 1842, Dr. Ch. W. Bell mentions, among the symptoms which occasionally predominated, jaundice, and adds in a note: "A kind of jaundice was common during the height of the epidemic." It was attended with slight symptoms of ague. "In the fever that I saw in the island of Karrach, in 1839, all died jaundiced."³ Jaundice, of various grades of intensity, is a well-known symptom of the remittent fever of the Mediterranean—Minorca, Sicily, and the Ionian Islands,⁴ where, as Dr. Craigie states, the countenance becomes gradually deeper and more dingy in colour, till it is almost brassy or orange-like; while the conjunctiva becomes yellow. The fever which prevails so extensively in various sections of Algeria, and which has been so well described by several of the medical officers of the French army of occupation, is often characterized by the symptom in question.⁵ The readers of Hippocrates cannot have forgotten the 62d and 64th aphorisms of section 4: "When jaundice supervenes in fevers before the seventh day, it is a bad symptom, unless there be watery discharges by the bowels." "When, in cases of fever, jaundice occurs on the seventh, the ninth, the eleventh, or the fourteenth day, it is a good symptom, provided the hypochondriac region be not hard." Did we not know, from the observations of modern physicians, that, in the remittent fever of Greece, jaundice is a common attendant, the above sentences of the father of medicine would establish the fact beyond dispute. It is, indeed, mentioned among the symptoms noticed by him in several of the cases reported in the *Epidemics*.⁶ Jaundice to this day constitutes a not unusual complication in the fevers of the Morea, where it was noticed, among others, by the physicians of the French army.⁷ The same symptom is a common attendant on the bilious remittent fever of Europe generally—France, Spain, Italy, Germany, Belgium, Holland, and England;⁸ and I need scarcely remark, that it is perfectly familiar to all who are acquainted with the same fever as it pre-

¹ Rankin's Report on the Pali Plague, p. 215.

² Marshall, Med. Topog. &c., of Ceylon, p. 138; Cameron, Edinb. J., lxxi. 73-4.

³ Brit. and For. Med. Rev., xvi. 561.

⁴ Cleghorn (4th ed.), p. 176; Irvine, Dis. of Sicily, p. 43; Burnett, p. 426; J. Davy, ii. 227; Craigie, i. 159.

⁵ Maillot, pp. 40, 150; Haspel, ii. 168; Boudin, p. 155.

⁶ Book I., Sydenham ed., i. 362; Littré, ii. 643; Book III., Syd. ed., i. 388-9; Littré, ii. 35.

⁷ Roux, Hist. Méd. de l'Armée Française en Morée, pp. 60, 61.

⁸ Sydenham, i. 210 (ed. of Syd. Soc.; Davis on Walcheren Fev., p. 23; Meli, Sulla Feb. Bil., p. 36; Pringle, pt. 3, chapt. 4, p. 172; Copland, i. 948, 984; D. Monro, Dis. of Army, ii. 73; Grant, Obs. on Nature and Cure of Fever, i. 232; F. Home, Med. Facts, p. 18; Nieuwenhys, Trans. of Provincial Med. and Surg. Assoc., iv. 68; Monfalcon, p. 275; Tissot, Epid. de Lausanne, p. 70; Finke, De Morb. Biliosi, p. 30; Rasori, Feb. Epid. de Genoa, p. 18; Wagler, De Morbo Mucoso, pp. 26, 83; Strack, De Morbo cum Petech. (1776), p. 145.

sents itself in the United States, and in other parts of America, north and south.

To this must be added that in chlorosis, a pale, earthy, leaden, pale green, and icteroid hue of the skin—amounting, in some cases, to a bronzed jaundice colour—has been noted from time immemorial and in all countries;¹ that one of the effects resulting from the action of animal poisons on the economy is a jaundiced, bronzed, and even sometimes mahogany discoloration of the skin;² that some of the vegetable poisons give rise to like phenomena;³ that jaundice, and other symptoms of yellow and miasmatic fevers are, at times, occasioned by several varieties of cryptogamic products;⁴ and that dark and yellow discolorations of the skin result occasionally from the action of vegeto-animal effluvia.⁵

The frequent absence of the discoloration in question in yellow fever has been noticed in both hemispheres. Everywhere the disease, in many instances, proceeds through its various stages, to recovery or death, without the manifestation of the phenomenon; and everywhere the circumstances under which it appears, the causes which serve to modify it, the periods at which it shows itself, the different phases it assumes, are similar. In this city, its absence, during our several epidemics, has been pointed out by, or may be inferred from the statements of Rush (iii. 68; iv. 12), Deveze (pp. 4, 36), Barnwell (p. 372), Jackson (p. 367), Currie (p. 31), and Wood (i. 301).

The same results have been obtained in Boston,⁶ New York,⁷ Middletown,⁸ Baltimore,⁹ Norfolk,¹⁰ Charleston,¹¹ Natchez,¹² New Orleans,¹³ and Savannah.¹⁴ A similar absence of jaundice, in a larger or smaller number of cases, has been noted in Europe;¹⁵ and, on consulting the writings of tropical physicians, we shall perceive that they have not been remiss in calling attention to the same fact.¹⁶

¹ Speranza, *Della Clorosi Commentario*, p. 21; *British and For. Rev.*, xiii. 332; Marshall Hall, *Cycl. of Pract. Med.*, i. 376; Desormeaux, *Dict. de Méd.* (1st ed.), v. 169; Ashwell, *Pract. Tr. on Dis. peculiar to Women*, p. 18, Am. ed.

² Fontana, i. 67; Celle, *Hyg. des Pays Chauds*, p. 89; Ed. Miller, *Works*, p. 54; *Ib.*, *Med. Reposit.*, ii. 413; Ferguson, *Recollections*, pp. 204–5; Hunter, *Dis. of the Army*, p. 156; Christison on *Poison*, p. 485; Hallowell, *Tr. of Col. of Phys.*, N. S., i. 396.

³ Miller, *Med. Rep.*, ii. 412, *Works*, p. 54; Rochoux, p. 79; Costa Sicre, p. 79; Salva, *Segundo año del Real Estudio*, &c. p. 142; Sauvages, iii. 112; Lemonier, *Mem. of Acad. of Sciences for 1849*.

⁴ Mitchell, *Crypt. Orig. of Fever*, p. 73.

⁵ Rochoux, pp. 80–2; Steva, *Observaciones Medicas*, &c., p. 49.

⁶ Warren, in Tytler, p. 502.

⁷ E. H. Smith, p. 124; Bayley, p. 96; Seaman, p. 10.

⁸ Tully, pp. 299, 300.

⁹ Drysdale, i. 132.

¹⁰ Valentin, p. 170; Archer, pp. 67–9.

¹¹ Irvine, p. 30; Shecut, p. 120; Wragg, *Charleston Journ.*, x. 80.

¹² Merrill, ix. 246; Hogg, p. 410; Stone, p. 557.

¹³ Barton, xv. 63.

¹⁴ Waring, p. 46.

¹⁵ Rochoux, p. 470; Audouard, p. 66; Arejula (*Fev. of Malaga*), *Edinb. Journ.*, i. 450; Pym, p. 5, *ib.* *Ed. Journ.*, xxxv. 41; T. Smith, *ib.* 42; Louis, pp. 173–246, 7, 8, 305, 6; Palloni, p. 9; *Ed. Journ.*, ii. 85.

¹⁶ Hume, p. 201; Desportes, i. 196; Pym, p. 76; Chisholm, p. 128 (1st ed.); Savarésy, pp. 279, 280, &c.; Pugnet, p. 359; Moseley, p. 429; Hunter, pp. 318, 319; Jack-

As regards the proportionate number of cases in which the jaundice occurs, we possess but few data upon which to form a positive conclusion. From all that we can gather on the subject, we may infer that the number varies in different places during the same or different seasons—being limited in some and considerable in others; that the same difference is found to occur in the same place, during different seasons; and, moreover, that the proportion varies according to the issue of the disease, and the rapidity of the attack.¹ Of 323 cases observed in the West Indies by Dr. Maher, jaundice did not manifest itself in more than 36, or 1 in about 9.5 (p. 850). After remarking that the yellow suffusion is not always present, Dr. Arnold adds: “Indeed, in the greater number of cases which I have seen, it did not exist at all” (p. 9). On other occasions, in the West Indies, it has been as often absent as present.² Madrid speaks of it as of unfrequent occurrence at the Havana (p. 31). In Dr. Barton’s Table of Symptoms in the fever of New Orleans, in 1833, jaundice stands as 6 against 59, in whom the skin was of the natural colour. Among 2,071 cases of the severe and mild forms of yellow fever, noted by Dr. Blair, 385 had yellow skin, being in the proportion of 18.54 to 100. At Natchez, in 1823, it was only a casual symptom, and rarely observed to much extent, except in the last stage of the disease.³ In Malaga, in 1804, it was not a common symptom.⁴ In 254 cases treated in the Roper Hospital in 1854, only 149 had jaundice.⁵

In other places, it has proved a more common symptom than in those above mentioned. Sir G. Blane remarks, in his work on the Diseases of Seamen, that jaundice was very commonly observed in the cases he saw, appearing not only in those who were seriously ill, but in some who were scarcely so (pp. 430–6–440). Inray found it invariably present (liii. 82). In Barbadoes, in 1817, it appears to have been very generally noticed.⁶ It was equally so in the epidemic described by Pugnet (p. 359). Lefort states that at Martinique, in 1825, jaundice appeared in almost every case.⁷ In 1817, at Charleston, it appeared in the great majority of the cases in a perceptible degree.⁸ In our epidemic of 1793, the disease was seldom unattended with yellowness of the whole, or of some portion of the surface. The eyes seldom escaped.⁹ The same result obtained in 1794.¹⁰ It would appear to have constituted an almost constant symptom in the epidemic of Leghorn in 1804,¹¹ and of Cadiz in 1800.¹²

The proportion in which jaundice occurs in the same place at different times, has been known to differ very materially. Dr. Rush states that the

son (Tr.), pp. 251, ii. 276; Gillkrest, p. 273; Musgrave, vii. 135; Madrid, p. 31; Osgood, pp. 11–14; Rufz, pp. 16–52; Ralph, ii. 73; Lempriere, ii. 85; Dubreuil, viii. 322; Dickinson, p. 50; Bally, 2–7–234; Dancer, p. 83, note; Maher, pp. 849–872; Wilson, p. 188; Levacher, p. 85; Blane, p. 430, &c.; Arnold, p. 9; Blair, p. 78.

¹ Rufz, pp. 16–53.

² Gillkrest, p. 273.

³ Merrill, ii. 226.

⁴ Arejula, Ed. Journ., i. 450.

⁵ Wragg, op. cit., p. 30.

⁶ Ralph, ii. 73.

⁷ De la Saignée, p. 581.

⁸ Dickson, iii. 263.

⁹ Rush, iii. 68.

¹⁰ Ib., iii. 211.

¹¹ Palloni, p. 9.

¹² Arejula, p. 175, and Ed. Journ., i. 450.

yellowness was more universal, but more faint in 1794 than the year preceding (iii. 211). In 1797, it again ceased to be so universal (iv. 12). Hunter, long ago, remarked that in the West Indies it was more frequent in some seasons than others (p. 319). This is exemplified by facts already mentioned. We have seen, on the testimony of Lefort, that in Martinique, in 1825, it was almost invariably present; while, according to Rufz, the same symptom never, or very seldom appeared among those who recovered and constituted the larger proportion of cases. It was a common symptom at Charleston in 1748,¹ and in 1817.² We may infer from various indirect statements of Dr. Irvine that it was not so in 1820. We have seen that the symptom did not prevail extensively at New Orleans in 1838. In 1839, we learn it was of very frequent occurrence (*Report*, p. 324). Sir James Fellowes informs us that at Cadiz, in 1804, the yellow colour of the skin was not so commonly encountered as it had been in the epidemic of 1800 (p. 198).

To this it may be added, that the frequency of jaundice varies at different periods of the same epidemic. In some seasons it is more common at an early period, and gradually becomes less so as time advances; in others, the reverse is the case. The early cases rarely present the icteric coloration, at least during life; while later, the skin becomes yellow in almost every instance. The latter observation was particularly made during the epidemic of Cayenne, in 1850-51.³

As a general rule, jaundice may be stated to present itself much less frequently in those who recover, than in those who die. Dr. Rochoux remarks that in the West Indies it is absent in at least one-half of the cases that recover; whereas, among fatal cases, scarcely one out of one hundred occurs, in which yellowness may not be discovered in the eyes, on the face, neck, or chest (p. 307.) Rufz states, that at Martinique, in 1838-9, jaundice occurred in all fatal cases, but was observed by him only twice in 1838, and in no instance the next year (pp. 16-52). According to Moseley (p. 429), those who recover seldom have the jaundice, and it is present, though not always, in those who die. Blair, while remarking that it was absent in a large number of mild cases, states that it was present in every bad one, and was certain to show itself after death (p. 78). A similarly striking difference has been observed in Gibraltar in 1828, where, according to the facts recorded by Louis, jaundice was a very frequent attendant on fatal cases. Not so, however, in others. "It was not observed frequently in those who recovered; according to our tables, in a little less than a third of these patients. And this proportion is in reality too great, since the facts which we analyze have been selected, as I have already observed, by our professional brethren, as the most interesting on account of their severity, or on account of other important circumstances" (pp. 246, 7, 8).

This, it may be remarked, is in direct opposition to the assertion made by Rochoux (pp. 470, 569), that jaundice is absent in a great number of fatal cases in the yellow fever of temperate regions; while, on the contrary, it

¹ Lining, p. 421.

² Dickson, see *ante*.

³ Ann. Mar., 1852, viii. 178.

appears in all those who recover—thereby differing essentially from the fever of tropical regions. But the statement of Louis is sustained by the testimony of most writers in this country, where jaundice is much more frequent among fatal cases than among those that recover.¹

The occurrence of jaundice varies in point of frequency according to the rapidity of the cases, whether these terminate in recovery or death. In this country, when the disease is cut short, by art or the efforts of nature, in a short time, or when it carries off the patient before the third day, yellowness is of rare occurrence.² Statements of the same kind are found, so far as concerns the West Indies, in the writings of Dr. R. Jackson,³ and others. The jaundice was fainter here in 1794 than in 1793, yet the disease was more malignant.⁴

After what has been said of the more frequent absence of jaundice in cases that recover, we must naturally be prepared to find it more frequently in the malignant form than in mild cases of the disease—death being more usually the result of the former. Such, however, is the case, whether they terminate favourably or fatally.⁵ Lempriere (ii. 82) says that jaundice is more frequent in the hybrid than in the true continued endemic, shows itself earlier, and spreads in a much shorter space of time. The former is the more violent and malignant form.

Some difference exists, in various places or seasons, as also in various cases during the course of the same epidemic, in regard to the particular hue of the skin—the shades varying from a pale straw or muddy to a dark-reddish, or brown mahogany, or copperish colour; embracing, between the two extremes, the different modifications of yellow, orange or saffron—light and deep. It is generally of a uniform, but sometimes of a mixed and variegated character. These various shades of colour do not appertain individually to any special region, but have each been found to occur, under particular circumstances, in all parts where the fever has prevailed—sometimes at different seasons, at others, during the same season. Such has been the case in this city, where, however, the deep tints mentioned seem to have more generally been observed. Thus, Dr. Rush represents it as being sometimes of a deep orange (iii. 211). In many cases, it was composed of such a mixture of colours as to resemble polished mahogany (*ib.*). In others, again, it was of a clay colour (iii. 68). Dr. Monges represents it as being most commonly of a yellowish-brown, or even mahogany tinge (ii. 57). Dr. Wood states that it varies from a deep lemon to an orange or bronze colour (i. 301). Dr. Currie remarks that, in 1793, the yellowness, which at first was faint and partial, assumed a deep orange or saffron colour, and extended itself over the whole surface of the body, so that the patient resembled one with an obstinate and inveterate jaundice; next, it became of a deep tawny or dull copper colour; and, finally, of a “deep dusky yellow and purple colour, resembling blood settled in a bruised

¹ S. Jackson, Barton, Palloni, Tully, Irvine, &c.

² Currie, p. 31.

³ Sketch, i. 73–74.

⁴ Rush, iii. 211.

⁵ Desportes, i. 196; Lempriere, ii. 85; Pym, Edinb. Journ., xxxv. 41; Smith, *ib.*, p. 42; Savarésy, p. 279–282; Hunter, p. 318.

part" (pp. 23–27). In 1820, according to Dr. Jackson, the skin in some cases assumed a dark, mottled, and dusky hue, giving the complexion a mahogany tinge. In others, the skin became of a yellow colour, of greater or less intensity (p. 53). The epidemics of 1853, as well as the less extensive and important visitation of 1854, afforded instances of the same kind.

In New York, it has varied considerably—being sometimes of a pale lemon colour; or of a greenish, mottled, or bruised appearance; or darker, resembling a vegetable stain, or a dead body which has begun to putrefy. In other cases, it has resembled a deep orange or saffron colour.¹ Dr. Hosack remarks on the subject, that the yellowness of the skin in this disease partakes, in most cases, of the colour of the orange mixed with the reddish hue of copper—differing in that respect from that of bile, which is a golden yellow (p. 353). Dr. Smith (E. H.), in his account of the epidemic of 1795, describes the skin as being sometimes exceedingly yellow—even tawny (p. 124); and Dr. Pascalis speaks of the skin exhibiting, in 1821, a "*couleur mélangée d'un rouge foncé et jaune donnant à la figure une couleur d'acajou pâle.*"² In other cities of this country, the skin has presented the same diversities of colour; often, perhaps mostly, with an equal disposition to assume the darker hues, yellow, copperish, mahogany, saffron, deep orange, muddy dun yellow, bronzed, livid, blackish, &c.³ In Europe, the skin has, as it would seem, occasionally assumed a bright-yellowish tinge, or a pale citron or lemon colour.⁴ But there also the deeper shades were more frequently observed, especially at a more advanced period; the surface being of a deep ochre yellow, sunburnt, dingy yellow, greenish, blackish livid, intense yellow, reddish, leaden, dusky, marbled, saffron, or brownish hue, with or without patches.⁵ Nor shall we find the fever of tropical regions different in that respect; for, while some writers describe the skin as being of the colour of an unripe lime, of a dirty parchment, melon, orange, pale lemon, or citron-like tinge, the same and other writers also variously describe it as being, in many cases, of a deep-yellow, or true orange, or crocus, chrome, ochre, gamboge colour; of a dingy brown; or as presenting a mixture of livid, greenish, and dirty yellow; or the "colour of an American Indian;" marbled; of a mahogany colour; of a parti-coloured aspect, in which the principal hue is leaden or black; covered at times with dark patches of various sizes, surrounded with a yellowish or greenish circle, and resembling those resulting from contusions.⁶ In the fever which prevailed on board the French

¹ Townsend, p. 150.

² Marseilles Journ., v. 140.

³ Revere, iii. 324; Jameson, vi. 446; Hill, v. 90; Ticknor, iii. 227; Lining, ii. 421; Gros, p. 10; Girardin, p. 56; Thomas, p. 54; Cartwright, ix. 11; Shecut, p. 120; Kelly, p. 378; Harrison, p. 136; Tully, p. 299; N. O. in 1820, p. 9; Valentin, p. 177; Munson, xiv. 181; Dickson, p. 348.

⁴ Gillkrest, p. 271; Burnett, pp. 9, 47; Amiel, in Johnson, p. 263.

⁵ Jackson, pp. 74, 91; Arejula, pp. 160, 419; Gillkrest, p. 273; Boyd, in Johnson, p. 300; Rayer, p. 21; Burnett, p. 9; Gilpin, v. 322; Audouard, pp. 60–63; Caisergues, p. 170; Berthe, pp. 81–88; Pariset, Obs., p. 30; Pariset, Rep., p. 432; Palloni, Edinb. Journ., ii. 84; Sir J. Fellowes, p. 266; Rochoux, p. 470.

⁶ Frost, xiii. 3; Wilson, pp. 189, 223; Pym, p. 230; Lempriere, ii. 62, 63; Savarésy, pp. 276–280; Pugnet, pp. 356, 357; McArthur, pp. 346, 347; Imray, liii. 80–82; Ralph,

war-steamer "Gomer" in 1843, the mahogany-red colour of the skin, so common in other epidemics, was not observed.¹

In whatever place or region the jaundice of the yellow fever is made the subject of observation—whether in this city, in other parts of the United States, or elsewhere—we find that the nature and shade of the hue it assumes varies according to the period or stage of the disease, the grade or variety of the latter, the constitution or temperament of the patient, and the natural colour of the skin. At first more or less faint, it rapidly, but generally in a gradual manner, becomes darker as the case progresses, and continues so to do till the close of the attack. We have seen, from Currie's account of the fever of 1793, that such was the mode of progression here on that occasion. The history of all our other epidemics will show that, at every return of the disease, a like observation has been made; and it would not be difficult to prove that the same holds good of the fever as it shows itself elsewhere, whether in temperate or tropical regions. In the mild grade of the inflammatory form, the jaundice assumes the lighter shades, while the deeper or darker discoloration shows itself in the malignant or congestive forms of the disease.² The rule, however, is not absolute. Those who have observed the fever of this city have not unfrequently had occasion to find the most malignant forms of it characterized by a pale lemon or dirty parchment-like jaundice. In that variety of the disease which, by Pym and some others is designated by the name of Bulam fever, and which is of the most malignant character, the jaundice, both in Europe and in tropical regions, has, on the several occasions referred to, assumed the pale lemon colour, while in the milder forms of the disease the skin was of a very deep yellow tinge. It may be doubted, however, whether this circumstance, on which Pym lays great stress³—serving as it does, according to him, to establish a line of demarcation between these two fevers—it is doubtful, I say, whether the true Bulam fever was, in the epidemics witnessed by Pym, as universally marked by the kind of jaundice alluded to as he pretends; for others, who observed the disease in the same or neighbouring places, describe the skin as having usually become of a livid yellow, very yellow, dark yellow, or dingy yellow hue.⁴

But if there may be some doubts as to the correctness of Sir William Pym's statement relative to the universality of the occurrence to which he refers, there can be little reason to refuse our assent to the fact, not only that a pale jaundice is everywhere often an attendant on malignant cases of yellow fever, but that it has, on some occasions, been so very generally, if not

ii. 73; Musgrave, ix. 124; Osgood, p. 12; Bally, p. 229; Caillot, p. 19; Chisholm, i. 176, 199; Stewart, Med. Reg., iii. 187; Frost, xiii. 32; R. Jackson, Sketches, 57, 70–76; Ib., Treatise, p. 261; Bancroft, p. 47; Rochoux, p. 309; Blair, p. 79.

¹ Joubert, Ann. Mar., 1844, ii. 964.

² Wilson, p. 233; Osgood, p. 12; Barton, xv. 36; Townsend, p. 120; Kelly, p. 379; Girardin, pp. 34, 56; Fev. of Cayenne in 1850–1, Ann. Mar. 1852, viii. 178; Bancroft, p. 56; R. Jackson, pp. 183–5; Gillkrest, Moseley, &c.

³ Pp. 4, 5.

⁴ Arejula, p. 150; Fellowes, p. 266; Gilpin, Med.-Chir. Tr., v. 312.

uniformly. At Antigua, in 1817, as we are informed by Dr. Musgrave, in the mild forms of the disease which attacked natives or assimilated constitutions, the yellowness was generally deep, approaching to orange. In new comers, who had the disease in a concentrated form, "a pale lemon more represented the tint."¹ Dr. Stone, in his account of the fever of Natchez in 1848, says: "When the yellow colour appeared or increased towards or after the period of calm, a dark yellow was not an index of as much danger as a light lemon; indeed, the latter never appeared before this time, and only in the severer cases; whereas, a deep yellow was sometimes presented from the commencement, and was of no material consequence."² To this it may be added, that supposing those mild cases in which the skin assumed a deep yellow colour to have all been cases of true yellow fever—which is doubtful—the effect may be ascribed, in the West Indies more particularly, to the peculiar natural colour of the skin. In New York (1822), individuals of a fair complexion presented a surface of a pale lemon colour, or of a greenish, mottled, or bruised appearance. In other cases, the colour was darker, resembling a vegetable stain, or having the appearance of a body in an incipient stage of putrefaction; while the skin, in persons of a sallow complexion, became of a deep orange or saffron colour. In Antigua, where, as just seen, jaundice of an orange hue predominated, the complexion of the natives and assimilated was doubtless sallow; while new comers may reasonably be supposed to have had, for the most part, fair skins. The same remarks will hold in regard to the cases observed by Sir W. Pym.

The jaundical discoloration of the surface in yellow fever, is not, usually, as we have seen, an attendant on the early stage of the disease. It makes its appearance in cases characterized by febrile reaction, after the decline or cessation of the fever; and, in instances of a different kind, after an interval of longer or shorter duration from the onset of the attack—in all instances, the earliness of its appearance being proportioned to the severity of the disease. As a general rule, we may state, that it rarely, though sometimes, appears before the third day, more generally on that or the fourth day; often about the fifth or seventh, sometimes at the approach of death, after which it usually increases. These results, which were obtained in this city, at every return of the disease,³ have never failed in other parts of this country and other temperate regions, as also in tropical climates;⁴ the difference being

¹ Med.-Chir. Trans., ix. 124.

² N. O. J., vi. 558-9.

³ Rush, iii. 68; Deveze, p. 25; Currie, p. 23; Barnwell, p. 371; Ffirth, p. 28; Cathrall, p. 29; Wood, p. 301; Caldwell, p. 84; Jackson, p. 53.

⁴ Lining, p. 298; Drysdale, i. 132; Dalmas, p. 8; Valentin, pp. 167-171; Gros, p. 10; Girardin, p. 34; Harrison, p. 132; Thomas, p. 84; Archer, v. 67; Hill, v. 90; Dickson, iii. 254; Shecut, p. 120; Moultrie, p. 3; Ticknor, iii. 227; Cartwright, ix. 11; Baxter, xxi. 3; Townsend, p. 182; Smith (of Galveston), p. 502; New Orleans, 1820, pp. 9, 10; Bayley, p. 95; New Orleans, 1839, p. 322; Kelly, xiv. 377; Ticknor, iii. 227; Stone (at Woodville), ii. 181; Louis, pp. 246, 7, 8; Arejula, p. 161, and Ed. Journ., ii. 448; Fellowes, 52, 202; Audouard, p. 58; Pym, Ed. Journ., xxxv. 41; Burnett, p. 9; Berthe, p. 81; Gillkrest, li. 271; Amiel (in Johnson), p. 263; Palloni, p. 4; Pariset, pp. 386-

noticed in equal or various proportions during the same epidemic, or at various times in the same or different places. In other instances, jaundice shows itself only after death,¹ and, in some instances again, after the recovery of the patient, or even during convalescence.²

We have no means of ascertaining with any precision, by a reference to tabular records of cases, the days upon which the jaundice has most frequently made its appearance in the fever of this city. So far as I can ascertain at present, Dr. Blair, of Demerara, is the only one who has attended carefully to the subject. According to him, of 385 cases (out of 2,071) who had yellow skin, the symptom showed itself—

On the 1st day	8 times.	On the 8th day	18 times.
“ 2d “	16 “	“ 9th “	7 “
“ 3d “	46 “	“ 10th “	8 “
“ 4th “	89 “	“ 11th “	15 “
“ 5th “	86 “	“ 14th “	3 “
“ 6th “	66 “	“ 15th “	1 “
“ 7th “	30 “		

The yellow skin is closely associated with black vomit as to the time of appearance; but, as Dr. Blair has remarked, in reference to the fever of Demerara, the former, here and elsewhere, is generally the antecedent. Thus, in 139 ascertained cases, observed by that intelligent writer, the jaundice preceded the black vomit in fifty-one instances, appeared simultaneously in forty-six, and succeeded it in forty-two (pp. 82, 83).

The yellow discoloration of the skin is usually first perceived on the conjunctiva, at the outer angles of the eyes; in some instances, at the corrugations of the forehead, or angles of the jaws. It is sometimes first seen at the roots of the alæ nasi, the face, or neck. But, in whatsoever spot it may show itself originally, the jaundice generally extends. The whole of the part becomes tinged. The change follows the line of the large vessels of the neck. It extends next, sometimes with great rapidity, by bands or patches, which, coalescing, cover the chest. It proceeds also downward along the back, and finally diffuses itself over the whole body. It imparts, by mixing

397, and Obs. p. 30; Velasquez, p. 10; Boyd (in Johnson), p. 300; Dufour, iv. 51; Rochoux, p. 458; Wragg, p. 80.

R. Jackson, Sketch, i. 71; Bancroft, p. 34; Saverésy, pp. 273–9–282; Lempriere, ii. 62–85; Moseley, p. 437; Hillary, p. 149; Bruce (in Lind), p. 278; Frost, Med. Rep. xiii. 32; Peixotto, New York Med. and Surg. Journ., i. 413; McArthur (in Johnson), p. 346; Osgood, p. 11; Caillot, p. 18; J. Clark, p. 9; Maher, Ann. Mar., 1838, p. 849; Pugnet, p. 356; Imray, p. 82; Rufz, p. 15; Pym, p. 230; Bally, p. 229; Dyott, p. 1003; Pinckard, ii. 227–263; Wilson, pp. 22, 23; Musgrave, ix. 124; Rochoux, p. 267; Vatable, p. 345; Gillkrest, ii. 271–5; Lefoulon, p. 371.

¹ Moultrie, p. 5; Shecut, p. 120; Rush, iii. 89; Deveze, p. 58; Audouard, p. 66; Dalmas, p. 14; Wood, i. 301; Dancer, Med. Ass., p. 83, note; Harrison, ii. 136; Gillkrest, ii. 274; Vatable, p. 346; Arnold, p. 15; Maher, pp. 850, 872; Rufz, p. 16; Rochoux, p. 458; Bancroft, p. 34; Berthe, Mal. de l'Andalousie, p. 81; Bally, pp. 234–37; Fellowes, pp. 55, 60; Stone, New Orleans Med. Journ., ii. 181; vi. 555.

² Stone, New Orleans Journ., vi. 557.

with the redness which it replaces, and under which it may, at the outset, be discovered by pressing out the blood from the capillaries with the finger, a somewhat singular, and certainly peculiar appearance. This is more especially noticed about the face and neck, where the appearance consists in a yellow or dingy flushing, with seeming fulness. These varieties, in regard to the location of the first appearance of the discoloration in question, and the extent of its diffusion, have been noticed in our several epidemics. In 1793, according to Dr. Rush, it sometimes appeared on the neck and breast, instead of the eyes. In one of his patients, it discovered itself, first, behind the ears and on the crown of the head, which had been bald for several years. The eyes, however, seldom escaped a yellow tinge (iii. 68). Much the same statement, as regards the point of origin and mode of progression, is made by Deveze (p. 25), Currie¹ Caldwell,² Barnwell,³ Wood (i. 301), Jackson (pp. 52, 53), and others, who have witnessed our yellow fever—a statement confirmed by the results of observation made during the epidemic of 1853. Similar observations have been made over and over again in every place where the disease has prevailed—whether in temperate regions,⁴ or within the tropics.⁵ To this it may be added, that the discoloration of the skin, though often spreading extensively, and pervading every part of the body, remains in some cases partial, affecting only certain parts—most commonly the eyes—sometimes, with these, the face or neck, or trunk; a phenomenon attendant generally on mild cases.

The jaundice penetrates deeply, colouring the adipose matter, the substance of the tissues and the periosteum of the bones, and tinges the sweat to such an extent sometimes, indeed, as to discolour the linen of the patient.⁶ It also manifests itself in the urine, which often contains the

¹ On Bilious Fever, p. 220.

² Fever of 1805, p. 84.

³ Diseases of a Warm and Vitiating Atmosphere, &c., p. 371.

⁴ Bayley, Fever of New York in 1795, p. 95; Report of Fever of New Orleans in 1820, pp. 9, 10; *Ib.* in 1839, p. 332; Drysdale, Med. Museum, i. 255; Dalmas, p. 8; Townsend, pp. 149, 150–172; Archer, Med. Record, v. 67; Tully, p. 299; Dickson, Phil. Med. and Phys. Journ., iii. 255; *Ib.* Essays, p. 348; Valentin, p. 167; Ticknor, North Amer. Med. and Surg. Journ., iii. 227; Gros, p. 10; Girardin, p. 34; Ash. Smith, xxv. 502; Moultrie, p. 4; Sheut, p. 120; Kelly, xiv. 375; E. H. Smith, p. 124; Wragg, x. 80; Burnett, p. 9; Amiel (in Johnson), p. 263; Palloni pp. 4, 5; Boyd (in Johnson), p. 300; Gillkrest, ii. 271; Pariset, Obs., p. 30; Velasquez, in same, p. 10; Pariset, Fever of Barcelona, pp. 386, 397; Jourdain, Ann. de la Méd. Physiol., v. 257.

⁵ Baneroft, p. 34; McArthur, pp. 346, 7; Pugnet, pp. 356, 7; Lempriere, ii. 62–85; Hillary, p. 149; Hunter, p. 72; Moseley, p. 437; Bruce, p. 278; Blane, p. 430; Jackson, Tr., pp. 259, 260; Imray, liii. 82; Osgood, p. 11; Jackson, Sketch, i. 71; Rufz, p. 16; Frost, xiii. 31–34–36; Peixotto, i. 412; Bally, p. 229; Wilson, p. 223; J. Clark, p. 9; Levaucher, p. 73; Davidson, viii. 248; Daneer, p. 83; Gilbert, p. 66; Pym, p. 230; Dyott, p. 1003; Maher, p. 849; Roehoux, p. 267; Lefoulon, p. 104; Evans, p. 246; Moreau de Jonnes, p. 104; Joubert, Ann. Mar., 1844, ii. 967; Vincent, p. 26; Jolivet, p. 10; Fev. of Cayenne, p. 167; Catel, Ann. Mar., 1844, iv. 225; Finlay, p. 15; Bone, p. 5; Hume, p. 199; Grant, p. 33; Carter, p. 6; Anderson, p. 6.

⁶ Rush, iii. 211; Wood, p. 301; Jackson, p. 53; Dalmas, p. 14; E. H. Smith, p. 124; Arejula, p. 419; Gillkrest, pp. 273–5.

colouring matter to which the peculiar hue of the skin is due,¹ and it has been remarked, that in scrofulous sores, the curdy discharge is generally tinged.² Dr. Rush mentions a case he saw in 1794, in which the patient tinged his sheets of a yellow colour by night-sweats many weeks after his recovery. In another case, there was an exudation from the soles of the feet, which tinged a towel of a yellow colour (iii. 211). Let it be remembered, in addition, that yellowness is observed in the blood before it shows itself on the surface, or in the eye; and this, both in arterial and venous blood, when the jet impinges against the side of a white dish. It is also seen around the edge of a blistered surface before being visible elsewhere on the skin.³

Jaundice is often of short duration, yielding readily and quickly, as is well remarked by Dr. Wragg, of Charleston, whose observations on this subject coincide with those made here, to the advance of convalescence. In some, when intense in degree, and general in extent, it is tedious in duration. In protracted cases, it sometimes clears away slowly and gradually, so that the patients are still affected with it long after every other trace of disease has disappeared, the bile appearing to pass off entirely by the kidneys. In others, jaundice disappears in a few hours, the skin becoming changed in hue between the morning and afternoon visits so as to leave the patient scarcely recognizable. "In those cases," Dr. Wragg says, "I remarked that this change was synchronous with the discharge from the bowels of a dark, tenacious, inodorous matter in greater or smaller quantity. This matter resembled pluff-mud, and sometimes was moulded into the form of the intestine, passing away in long ribbon-like lengths." Dr. Wragg was not able to ascertain, positively, the composition of this matter by analysis, but believes it to be bile mixed with thick mucus.⁴

Some difference of opinion appears to exist as to the degree of importance that should be attached to the symptom under consideration in forming an estimate of the probable issue of the case. In reference to this point, jaundice may be examined relatively to the time of its appearance, the shade it assumes, as well as the degree, extent, and rapidity of its diffusion.

1. Viewing the jaundice of yellow fever as derived generally from the bile, and judging of its effects in that disease by what is observed in others, in which yellowness of the surface is the result of that cause, Dr. Bancroft states that a general yellow suffusion, according to his own experience, and that of many practitioners with whom he conversed, was of little importance in the fever in question; and that if, by many writers, it has been associated with extreme danger, it is only because "the excessive vomiting which had produced it, had also produced other more destructive effects." At the same time he regards as a fact, admitted by all practitioners, that other forms of jaundice—by patches—indicate extreme danger. As the former is the form

¹ Wood, p. 301; Jackson, p. 56; Wragg, p. 78.

³ Ibid., p. 78.

² Blair, p. 79.

⁴ Charleston J., x. 80.

usually mentioned in the early stages of the disease, it would result that, according to Bancroft, the early appearance of jaundice did not import danger. Other physicians have advocated the same opinion in temperate and tropical regions. Doughty informs us, for example, that he often saw the bright yellow suffusion in the skin, both in Europe and in Jamaica, shortly after the attack, "and never found it of dangerous import, but always the contrary." Audouard, who saw the fever in Barcelona in 1821, did not consider the jaundice of the first stage as indicative of fatal issue; while that which comes on later he viewed as a symptom of a very serious character (p. 225). Nearly a century before, Towne appears to have been impressed with a similar opinion, regarding as he did the regular crisis of the disease, "as generally discovering itself by a suffusion of the bile all over the surface of the body about the third day, and maintaining that "the saffron tincture is frequently observed in the space of twelve hours after the attack, if you carefully inspect the coats of the eyes;" and that "the sooner it appears the more encouraging is the prognostic, if the intention of nature be not perverted by the preposterous use of cordials and alexipharmics" (pp. 23, 24). Dr. R. Jackson also (i. 182, 3), speaking of the yellow suffusion of the eye, as in jaundice, remarks that it indicates a crisis—sometimes favourable, sometimes fatal—and that the bright or brilliant yellow is ambiguous in most cases.

Whether the disease, to which such statements refer, must be viewed as identical with the one forming the subject of our present inquiries, I shall not stop to examine at large. But though unwilling to do so, I may be permitted to express a doubt on that point; because, while it is well known that the jaundice of bilious remittent fever is harmless, we find that the greater number of the writers cited believe in an identity of these with the yellow fever, properly speaking; and may well be supposed to have had in view, while expressing the opinion in question, cases which other physicians would consider in a very different light. But the principal reason for entertaining the belief is, that by the larger number of authorities it is maintained—and the experience obtained in this city, as elsewhere on this continent, may be adduced in their support—that the early appearance of jaundice is indicative of danger; and that this danger increases in proportion to the earliness of its accession. Thus, Dr. Rush remarks, in allusion to the epidemic of 1793, that the early appearance of yellowness always denoted great danger (iii. 68). So does also Dr. Caldwell: "A yellowness of the eyes and skin is considered by some practitioners as a favourable symptom, provided it occurs early in the disease. In the epidemic of 1805, this was not the case" (p. 58). A like observation as to the danger of the early appearance of jaundice has been made by other writers on the disease of this city.¹ According to Dr. Monges, the jaundice, when it appeared before the fourth day, was an unfavourable—indeed, a fatal sign. Barnwell regarded it as generally a mortal sign when it appeared before the seventh day. State-

¹ Currie, p. 38; Nassy, p. 26.

ments of similar import are to be found in other American writings. Lining long ago remarked, that when the jaundice occurred on the second day, the patient generally died on the fourth (p. 429). In 1817, at New Orleans, the appearance of yellowness before the sixth day was almost always a fatal symptom. Bayley (p. 96), Irvine (p. 30), Brown (p. 12), Kelly (p. 380), Archer (p. 67), A. Smith,¹ in this country, and Palloni, in Leghorn (p. 8), considered an early jaundice as highly unfavourable. Pym,² Gillkrest,³ in Gibraltar, viewed it in the same light when it came on at the close of the third day. So did also Velasquez, at Seville, who entertained fears of it when it appeared prior to the seventh day (pp. 10, 12). Pariset, at Barcelona, found it of bad omen before the third day (p. 452). Even Arejula, who affirms that in Malaga, jaundice was not regarded as unfavourable when appearing at any period of the disease, over the whole body, admitted that it was safer as a prognostic sign after than before the sixth day.⁴ And if we push our inquiries on this subject a little farther, we shall find, that of the writers on the fevers of the tropics, some⁵ regard jaundice as of unfavourable or fatal omen before the seventh day; others, as Hillary (p. 149), Osgood (pp. 11, 14), Lind (p. 283), do not consider it so before a later period—eighth or ninth day; that others, again,⁶ content themselves with regarding it as unfavourable in proportion to the earliness of its development; while a different set,⁷ specifying more precisely the limits within which it may be viewed with apprehension, state that it is surely indicative of a fatal termination when appearing on the first or second day, or up to the third, fourth, or fifth day. “The period,” says Mr. Maher, “at which jaundice makes its appearance during the course of the disease, is, according to the observations of Mr. Belot, a prognostic sign of great certainty. When it appears towards the third or fourth day, death will infallibly ensue; if it occurs towards the fifth or sixth day, the probability of a fatal issue is greater than that of recovery; finally, when it manifests itself only after the seventh day from the period of attack, recovery is almost certain.” Mr. Maher adds, that he had ample opportunities of verifying the correctness of these statements.⁸

By some, indeed,⁹ jaundice, especially when complete, and when associated with other unfavourable symptoms, is viewed as always denoting great danger; and Lempriere (ii. 265), goes so far as to say, that “few recover after the yellow suffusion has appeared in any considerable degree on the neck and parotid glands.” Dr. Warren, of Barbadoes, appears to have, a century

¹ Tr. of Acad. Med., p. 30.

² Ed. J., xxxv. 41.

³ Cyclop., ii. 273.

⁴ Fiebre Amarilla, p. 184; Fellowes, p. 60; Ed. J., i. 450.

⁵ Desportes, i. 199, 201; H. McLean, p. 103; Dariste, p. 179; Warren, p. 41; Gillespie, p. 57; Bruce, p. 278; Rufz, p. 216.

⁶ Savarésy, p. 287; Dubreuil, J. Univ., viii. 322; Lind, p. 278.

⁷ J. Clark, p. 18; Bally, p. 283; Pugnet, p. 359; Rochoux, pp. 267, 308, 553; Levacher, p. 89; Joubert, Ann. Mar. 1844, ii. 970.

⁸ P. 850, and N. O. J., x. 467.

⁹ Savarésy, p. 287.

ago, entertained an equally unfavourable opinion of this symptom (p. 15), and informs us that Towne retracted his former views before he died, and would willingly have called in all the copies of his work could he have found the means of doing so. Dr. Blair regards the yellow discoloration of the skin as always a sign of great intensity of disease, and in evidence of this states, that of 385 cases who, out of 2,071 of all grades, had yellow skin, 178 died; the proportion of cases in which the symptom appeared being 18.54, and the rate of mortality of the symptom 46.23 per cent. (p. 79). But to these opinions, or to that of our countryman, Dr. Irvine (p. 30), who regarded the jaundice as a certain precursor of the formation of black vomit—and, as an almost inevitable conclusion, as almost always a fatal sign—it is difficult to subscribe to the fullest extent, opposed as they are to the results of experience. That the appearance of jaundice must generally be viewed as a sign of serious import, especially when manifesting itself early, no one can deny. Under most circumstances, it seems to show that the cause has made a strong impression on the system, marking, as Dr. Musgrave says (p. 125), the intensity of the disease, and its degree of danger. But every physician who has seen the yellow fever, must have noted not a few cases, even of a serious character, and when the jaundice has appeared early, or attained considerable intensity, which nevertheless have terminated favourably. Examples of this kind are mentioned or referred to by competent authorities on the subject.¹ We know, also, that in the mild forms of the disease, which often, if not generally, end in recovery, jaundice is not unfrequently an attendant symptom. Such was the case here on several occasions, and at no time less markedly than in 1820.² In New York, two years after, as we learn from Dr. Townsend, mild cases presented themselves in which the only symptom was a peculiar greenish yellow tinge, which spread all over at the usual period (p. 150). We learn, indeed, from Sir Gilbert Blane—what, however, has not been confirmed by subsequent observers—that there is something contagious in this symptom. “It was observed,” he says, “in the Royal Oak, and Alcide, to extend to men who were but slightly indisposed; and, at the hospital, it spread to men in the adjoining beds, without imparting any malignity to their diseases.”³ Be this, however, as it may, so far as regards the early and middle periods of the disease, every physician—with few exceptions, perhaps—agrees in regarding jaundice, when it appears after the sixth, but more particularly the seventh or eighth day, as of little import, or even as a favourable or critical sign.⁴

2. From what has already been said respecting the connection between certain shades of discoloration of the skin, and the several varieties of forms and degrees of malignancy of the disease, it will readily be perceived that

¹ Rush, iii. 68; Arnold, pp. 16, 17; Bancroft, p. 48; Louis, p. 248; Ticknor, iii. 227; Vatable, p. 350; Savarésy, p. 297.

² Jackson, p. 61.

³ Blane, *Diseases of Seamen*, pp. 434–5.

⁴ Desportes, i. 201; Bruce, p. 278; Hillary, p. 149; Arejula, p. 450; Palloni, p. 8; Gilbert, pp. 72, 78; McLean, p. 95; Rufz, p. 16; Gillespie, p. 57; Maher, p. 850; Towne, p. 23; Fellowes, pp. 58, 60.

the former must furnish indications as to the probable issue of the case. For whichever shade may be associated with the malignant forms, which furnish the greater number of fatal cases, must necessarily be regarded as indicative of the greatest danger; and, as a general rule, it may be remarked—all things being otherwise equal—that the severity of the attack is proportioned to the deepness and general diffusion of the discoloration. Hence, we find Moseley stating that the universal deep and increasing yellowness of the skin, accompanied by an aggravation of other symptoms, is the immediate forerunner of death (p. 438). The experience of Lempriere (ii. 68) led him to nearly the same conclusions. The light lemon-coloured yellow of the eye, according to Dr. R. Jackson, is generally favourable; the deep yellow of the same part, with a shade of brown, like that of a Seville orange, is generally fatal; the bright or brilliant yellow is ambiguous. The same remark may be made as regards the colour of the face. If livid, as in sea-scurvy, or dark as the colour of mahogany, death may be expected; while, agreeable to the same high authority, jaundice of the surface, of the darker shades, is unfavourable (i. 183–5). Rochoux, in like manner, views the light yellow as the more favourable form of jaundice, and as indicative of recovery; the deep yellow—mixed or not with greenish brown—and the livid hue, as of dangerous omen, and indicating a fatal issue (pp. 268, 309). In 1822, in New York, “where the deep yellow tinge spread entirely over the trunk, the disease proved almost universally mortal.”¹ Gillkrest tells us, and the same has been very generally observed, that a shade of jaundice, similar to what occurs in patches in ecchymosed parts, or mottled skin, in which livid, light, olive, and ash-coloured patches, of all sizes, shade into each other, is a fatal symptom.² The clay-coloured appearance of the face, mentioned by Dr. Rush (1793) as occurring in the last stages of some cases, was universally regarded as prognostic of a fatal issue (iii. 68).

At the same time, cases not unfrequently occur—in some epidemics very generally—in which the light lemon colour of the skin is, as already stated, an index of greater danger than a dark yellow.³

3. Nor do the period at which the jaundice appears, and the particular hue it assumes, alone furnish us with indications as to the issue of the disease. Considerable importance is due to the degree of rapidity with which it spreads and increases in intensity. “When the danger is great,” says Dr. Rochoux, “scarcely have we discovered the first traces of the jaundice, before the whole surface is covered. It does not descend generally from the face to the neck, and thence to the chest and inferior extremities. It involves simultaneously the whole body” (p. 308). The danger of this rapidity in the diffusion of jaundice has been at all times noticed; while the comparative innocuousness of the discoloration, and even the benefit resulting from it when it occurs gradually, have not been overlooked. Lining mentions among

¹ Townsend, p. 151.

² Cyclop. of Pract. Med., ii. 273; Bancroft, p. 48; R. Jackson, i. 185; Kelly, p. 380; Rochoux, p. 268.

³ Pym, p. 5; Musgrave, p. 125; Stone, pp. 557–8.

the unfavourable signs, the rapidly increasing yellowness of the skin and eyes (p. 430). "Sometimes," says Dr. H. McLean, "jaundice, which came on by slow degrees, seemed to remove all the febrile symptoms" (p. 95).

On the subject of the cause of the discoloration of the skin in the yellow fever, a considerable difference of opinion has existed, and continues still to exist, in this country and elsewhere. In his account of the epidemic of 1793, Dr. Rush ascribed the yellow colour of the surface wholly to the admixture of bile with the blood; and such also was the opinion of most of the physicians who witnessed the disease on that occasion.¹ The experience which that eminent physician obtained during the course of the next season, induced him to modify his views on the subject; and, while continuing to believe that to the cause in question must be ascribed the yellowness in those cases where the colour is deep and endures for several weeks beyond the crisis of the fever, he thought that "where it is transitory, and, above all, where it is local, or appears only for a few hours during the paroxysm of the fever, it appears probable that it is connected with the mode of aggregation of the blood, and that it is produced wholly by some peculiar action in the bloodvessels" (iii. 211). Since that period, the latter opinion, but extended to most cases and circumstances under which the discoloration may appear, has been very generally advocated among us.²

The two theories of the bilious and sanguineous origin of the discoloration of the skin in the disease before us, have not divided, and do not continue to divide, the medical profession among us only; they have both commanded attention from an early period, and to this day enumerate partisans. The former, agreeable to which the phenomenon is ascribed to a redundancy of bile, and the regurgitation and absorption of a part of it, either before its discharge from the liver, or after, owing to some obstruction to its passage into the duodenum—from spasm of the duct or other parts; gall-stones, or simple inflammation, and consequent thickening of the mucous membrane; from severe contraction due to violent vomiting and straining, forcing the fluid into the vena cava—or, as is more recently believed, to the retention in the blood of the constituent principles of the bile, arising from a deficiency of hepatic secretion and elimination, was long ago, and is still, supported more or less exclusively by competent authorities.³

It is founded—1. On the analogy and supposed identity of the yellow with other fevers in which jaundice is undeniably due to bilious admixture, from

¹ Barnwell, pp. 371–390; Currie, p. 31.

² Caldwell, p. 89; Mongee, ii. 61; Wood, p. 301.

³ Towne, p. 23; John Hunter, pp. 72, 135, 157; Saunders, *Structure, Economy, and Diseases of the Liver*, p. 233; Savarésy, p. 478; Lempriere, ii. 102; Bancroft, pp. 32–48; *Ib.*, Seq., p. 22; Trotter, i. 357; S. Custin, *Duncan's Med. Com.*, ix. 238; Moseley, p. 429; Dickinson, p. 128; Robert, *Guide San.*, i. 323; Catel, p. 12; Maher, p. 853; Arnold, p. 16; Osgood, p. 14; Chisholm, i. 176; Drysdale, i. 132; Louis, pp. 123–141; Girardin, p. 40; Rochoux, pp. 309, 507; Stone, vi. 578; Chervin, *Réponse à Guyon*, p. 44; Levacher, p. 91; J. Davy, *Notes to Blair*, pp. 85, 158; Lefoulon, p. 348; Archer, *Med. Rec.*, v. 69; Cassan, *Mém. de la Soc. Méd. d'Emulation*, v. 100; Blair, p. 161.

absorption or other causes. 2. On the diffusion of the yellowness. 3. On its peculiar hue, and the close analogy between the latter and the discoloration in ordinary jaundice, in which the effect is due to such absorption (in the event of the existence of obstruction), or to the retention in the blood of the constituents of the bile. 4. On the concomitant deficiency of the bile in the primæ viæ, and the presence of the fluid, or of its colouring matter, in the products of the emunctories—more especially in the urine, judging from the colour of that fluid, and its becoming greenish on the addition of an acid. 5. On the ordinary association existing between its presence and the act of vomiting, which often brings on a jaundiced condition of the skin. 6. On the occurrence of the same discoloration, from an analogous cause, in cases of disease arising from the action of animal viruses or mineral and vegetable poisons. 7. On the rapidity with which it often disappears during convalescence. 8. On the impossibility of referring the lemon or deep orange hue, which the surface often assumes, to any other cause. 9. On the frequent existence of inflammation of the gall-bladder, biliary ducts, and duodenum, as revealed on dissection; a condition of parts which often gives rise to jaundice, and can only produce the effect by obstructing the passage of bile. 10. On the peculiar condition of the liver, which puts an impediment to the secretory functions of that organ, and, preventing the elimination, through its agency, of the constituent materials of the bile, causes it to be thrown on the tissues. 11. On the fact of the pre-existing discoloration of the serum of the blood, which discoloration arises from the presence of the constituents of bile.

Such a discoloration, says a recent writer, must not be ascribed to the yellow transition of the general ecchymosis, for it takes place at the same time with the latter, and does not succeed it; while, again, it does not occur in other diseases in which ecchymosis presents itself—as in cholera. If the yellowness was the effect of ecchymosis, the skin should first be dark; then it should become yellow, as the ecchymosis disappears. It must depend on some other cause; it exists alone sometimes, and is complicated with effusion of dark-coloured blood, for the production of the bronze and blackish yellow alluded to. Finally, the colour disappears in milder cases, when the skin is of a pale saffron hue, without passing through those gradations observed on the surface during the clearing of sanguineous transudations and ecchymotic extravasations.¹

The opinion which ascribed the discoloration of the surface to a particular condition of the cuticular circulation is very far from having originated with Dr. Rush. It was warmly supported, more than a century ago, by Warren, in his *Treatise on the Malignant Fever of Barbadoes* (p. 11), in a passage which embodies many an argument adduced as original in more modern times. “This yellowness, I am persuaded, chiefly arises from a more complete colliquation or dissolution of the red globules of the blood into a yellowish serum, which will naturally soon give that tincture to the whole skin. The same is often observable on human bodies soon after bites of

¹ Levacher, p. 93.

some poisonous serpents, or other venomous animals; and, in such cases, it cannot with any reason be supposed to proceed from a suffusion of bile, but rather from a colliquation, and perhaps a gangrenous diathesis of the sanguineous mass, occasioned by the force of the deleterious venom that had been infused into it. What is observed every day in all common bruises of flesh, may serve somewhat further to illustrate the matter; for here, when the texture of the extravasated blood begins to loosen and dissolve into a liquid serous consistence, a very visible yellowness appears in and about the part; but this soon goes off again, when the matter is fully absorbed back into the vessels, where it commits no hurt, but is readily overcome by the force of nature, as the quantity of such dissolved blood is small, and at the same time very innocuous. I do not, however, deny, but that through a great propensity and straining to vomit, some quantity of bile may be thrown into the blood; but then I must observe, that the yellowness of this distemper I am speaking of, very frequently shows itself when there has been no vomiting or retching at all, or scarce any sensible sickness of the stomach; for the truth of which I can appeal to many."

It has been since advocated in the explanation of the discoloration under the various circumstances and contingencies in which it presents itself, by a large number of respectable and competent authorities;¹ while, by others,² it is admitted that, though the jaundice of this form of fever is due to the ordinary agencies which produce that symptom in other complaints, yet some forms of coloration—appearing in the malignant form of the disease, or in the last collapse or putrid stage of every form—depends on the other cause mentioned, or on both combined.

This opinion, agreeable to which the symptom is the result in part, at least, of a yellowness of the serum arising from a colliquation or dissolution of the red globules; its separation from these, and admixture with their colouring matter and subsequent effusion under the cuticle; or, in great measure, of an *error loci* of the diseased globules in the white vessels, or the cellular tissue, as in ecchymosis; in other words, of the existence of a congestive state of the sub-cuticular capillary network, akin to that occurring in the mucous membrane, and giving rise to hemorrhages; this opinion, I say, is supported on the following grounds:—

1. The discoloration does not always appear first in the eyes, as is the case in jaundice; but shows itself as frequently, if not more so, in other

¹ Hillary, p. 150; Blane, p. 411; Thomas Clark, p. 9; H. McLean, pp. 32, 33–82; Pugnet, p. 359; Kéraudren, pp. 5, 6; Vatable, p. 355; Guyon, p. 755; Stevens, p. 261; Ferguson (in Recol.), p. 205; Townsend, p. 180; Audouard, p. 202; Desmoulin, Journ. de Phys., July, 1823; Med. Recorder, v. 565; Waring, p. 46; Dariste, p. 132; Harrison, N. O. J., ii. 137; Michel, Charleston Journ., v. 747; Arnold, p. 37; Dickson, p. 349; Evans, p. 247; Williams, p. 28; Dickinson, p. 133; Valentin, p. 177; Johnson, on Tropical Climates, p. 81; Jos. Frank, Méth. de l'Inst. Clin. de Pavie, p. 1795; Copland, iii. 145.

² Moultrie, p. 13; Moseley, p. 438; Bancroft, p. 48; Osgood, p. 14; Savarésy, pp. 378–380; Levacher, p. 91; Chisholm, i. 176.

parts—the face, neck, &c., and in its progress usually follows the course of the large vessels.

2. It is often partial or in streaks—much darker in some spots than in others. It is a subject of common observation that the bronze or mahogany hue disappears when pressure is made with the finger, but slowly returns when the pressure is removed—a result which does not obtain in bilious jaundice.

3. The discoloration of the skin in the yellow fever sometimes, nay, often, does not make its appearance till after death, when all redness in the capillaries has subsided, and the skin is in a condition similar to that in which it is placed when death takes place, after a part has been severely contused. This appearance of jaundice after death, and its instantaneous development to such a degree as to impart a yellow tinge to all parts of the system, are incompatible with the idea of reabsorption; for the power by which this process is executed must be lost with the death of the parts concerned. With the extinction of life, there is cessation of all organic functions.

4. The rapidity with which this discoloration is diffused over the body, and with which it assumes a dark, often the darkest hue, is incompatible with the idea of its originating from the common cause of jaundice, which, whether arising from ordinary morbid conditions of the liver or annexed organs, or connected with bilious remittent fever, is of slower progress in both these respects.

5. The biliary organs never, or very rarely, participate in a marked degree in the symptoms of yellow fever, which progresses, from first to last, without any increase in the quantity or alteration in the quality of the biliary secretion; and though jaundice is generally associated with a deficiency of bile in many cases of the disease attended with that symptom, the secretion is not invariably deficient in quantity, while the fluid, when secreted, remains generally of natural quality.

6. The colour of the surface in the yellow fever does not closely resemble that observed in jaundice or remittent bilious fever; or at least does so in exceptional cases only. The dingy, or pale muddy-yellowish tint, so common in the former, is found in neither of the others, nor indeed in any disease attended with yellowness due to a bilious derangement; while, in many instances, the colour approximates much more to that observed in a contused part, when the blood extravasated under the cuticle is being removed through the process of absorption. The dark brown, yellowish-red; the bronzed or mahogany hue of the skin; the violet or black tint; the marbled or clay-coloured appearance; the dingy gray and dirty olive, are all far removed from any colour resulting from effusion of bile or of the bilious colouring principle, and noticed in any form of jaundice; but are all readily explained on the supposition of an altered condition of the blood or bloodvessels of the parts—of a change produced by the extravasation or tardy circulation of serum of deep yellow colour, tinged with dissolved red or dark colouring matter, while the dark spots or blotches, discovered in different parts, are evidently and admissibly due to a sanguineous extravasation.

7. Sometimes, as remarked by Dr. Blair, ecchymosis of the eye, and yellowness of a circumscribed character, coexist and extend *pari passu*, the yellowness keeping one-eighth of an inch in advance of the ecchymosis. After the absorption of blood in convalescence, a deep orange colour is left in the blood spots. It may be added, that stains of an ochre colour in the brain and kidneys, in instances in which, it may be inferred, blood had been extravasated, were found, on chemical analysis by Dr. Davy, to depend on the presence of peroxide of iron, derived, no doubt, from the blood-corpuscles.¹

8. In jaundice, the urine is coloured by bile. Such is not the case in yellow fever, even at the time that the skin is highly discoloured. Cases doubtless occur in which the urine is found tinged; but these cases are rare, and are found connected with bilious jaundice, which sometimes, though not necessarily, supervenes in the course of the disease.

9. The jaundice of the yellow fever is connected with a deranged condition of the blood—imperfect power of coagulation, or a complete destruction of that power, with a yellow or even orange colour of the serum, mixed, in many cases, with a portion of red or dark colouring matter. This is associated with a relaxed condition of the extreme vessels, as evinced by the disposition to, and frequent occurrence of hemorrhage, from the mucous membranes and the skin itself. When, with these circumstances in view, we observe the peculiar coloration of the skin in yellow fever—a coloration inexplicable on the supposition of a bilious agency—we may readily attribute it to the cause in question.

10. The yellowness, which, as we have seen, first occurs in the eyes or face, succeeds to a red turgescence of the skin; as this turgescence, which is generally great, subsides, the dusky tinge is perceived, gradually spreading and becoming darker in proportion as the tone of the vessels diminishes and the alteration of the blood increases. The redness coincides with the existence of the florid appearance of the blood and activity of circulation, and the yellowness with the dark colour of the fluid and lentor of the circulation.

11. The effect of animal venoms, as well as of vegetable and mineral poisons, in inducing a discoloration of the skin not unlike that of yellow fever, may be adduced in further illustration of a sanguineous agency in the production of the latter; for the action of such poisons, in instances of the kind, is evidently exercised upon the blood, and the yellowness thereby produced is the effect of changes therein occurring, and not the result of a bilious admixture. That Mead (p. 9) and Fontana (i. 69), and after them many other writers,² attributed the yellowness consequent on the bite of the viper to this admixture, is well known. But, on close examination, it will be found that the main reason for entertaining this opinion is the fact of the existence of the yellowness, and, agreeable to some, the frequent occurrence in this case, and other instances of poisoning where the colour in question occurs, of violent vomiting and straining, by which the regurgitation of the bile is greatly promoted. On these grounds alone, however, such a view of the mode of production of yel-

¹ Blair, p. 79.

² Bancroft, p. 48; Levacher, p. 92.

lowness cannot be admitted; because none of these writers have proved that this coloration cannot be induced without the agency of bile; because none have tested the existence of that fluid, or its constituents, in the blood of persons labouring under the effect in question; and because yellowness is produced by the introduction of poisonous substances in the blood itself, is attended with evident morbid changes in that fluid, arises too rapidly to be ascribed to a secondary alteration and admixture of bile from absorption, and often is unattended with a degree of straining sufficiently great to justify the idea of regurgitation, or with any mechanical cause of obstruction whatsoever.

12. Yellowness of the skin, not very unlike that which occurs in some forms or stages of yellow fever, is an attendant on diseases in which the biliary organs and secretions are not necessarily or often implicated, but in which the blood is invariably morbidly affected—chlorosis, typhus, relapsing fever, &c.

13. The jaundice of yellow fever, when once formed, subsides much in the same way as the discoloration admitted to be produced by subcutaneous sanguineous effusion; and this subsidence is not promoted by those means which are found beneficial in jaundice from bilious suffusion.

14. Few physicians, at all experienced in the yellow fever, will refuse to admit that the dark ecchymotic appearance of the skin is due to the presence of blood under the cuticle. If such be the case, we can see no reason to deny a like origin to the mahogany and bronze appearance of the surface, gradually down to the dark, dusky, and finally dull pale yellow hue which it assumes, all of which are known to be producible by such a cause, and not to arise from bilious suffusion; particularly when we bear in mind that the ecchymotic appearance never exists alone, but is always combined with a jaundiced discoloration of the skin.

15. Jaundiced suffusion, in diseases in which it is the effect of bilious absorption, or of the retention and accumulation of the elemental constituents of the bile in the blood, and the want of their elimination therefrom, whether those diseases be of a febrile character or otherwise, is in itself a harmless symptom. In the yellow fever, on the contrary, it is, as we have seen, except under particular circumstances, and when it shows itself at an advanced stage of the attack, the harbinger of formidable danger, and, at an early period, the almost certain sign of a fatal issue.

Such are the facts and arguments that may be, and have been, adduced in support of the sanguineous origin and nature of the yellow discoloration of the eyes and skin in the yellow fever. If we extend the theory to the explanation of the symptom, viewed in a general way, and without reference to any particular modification of it, there can be no doubt that it possesses great advantages over the opinion which would ascribe the yellowness in question to a bilious suffusion, or to any peculiar condition of the hepatic functions. But, disposed as I am to regard it favourably, and to admit that it enables us to explain phenomena which the other theory cannot account for, I am not sure that, in the present state of our knowledge, we are justified in adopting it in all

instances, to the exclusion of the other, and to refuse our assent to the fact that the yellowness which occurs in the yellow fever is sometimes, if not often, connected with a particular derangement of the biliary organs, or a modified condition of the hepatic functions, and is therefore allied to ordinary jaundice. Those instances in which the skin is of a uniform saffron or lemon colour, and especially those in which the suffusion assumes the character of a critical change, or takes place at a very late period of the disease, when the powers of the system are already recruiting, or during convalescence, may, without risk, be referred to such a cause, and not to one which invariably implies the existence of considerable danger. Nor can we refuse to admit that the discovery of the constituents of the bile in the urine, in cases of yellow fever attended with jaundice—a circumstance analogous to that presenting itself in ordinary yellow discoloration—lends additional support to the opinion. Still more to the purpose are the facts, to which attention has been called, in which the perspiration imparts a yellow or saffron colour to the substances impregnated with it. In ordinary jaundice, the yellow colour of the serum of the blood, and of the crassamentum when covered with a buffed surface, is judged to be due to bile, not only from the appearance of the substances examined, and their effect in tinging linen, but also from the results of chemical tests; for though, from its combination with albumen, which defends it from the action of acids, it is difficult of detection, yet by Lassaigne the colouring matter has been found in the circulation, while others—Collard, Martigny, Lecanu, and Kane—have discovered the resin of bile in the blood of jaundice. Bile may, indeed, be detected in the blood, by the addition of sulphuric acid, which will cause the serum to change its yellow hue for the characteristic green colour of acid bile.¹ We have seen that the like presence of the constituents of the bile is discovered in the blood of yellow fever. It is found in the serum obtained from under the cuticles in blistered surfaces; this I have myself observed, on several occasions, in the presence of some medical friends. It was noticed, particularly, in a case which occurred at the Pennsylvania Hospital in 1854, where a teaspoonful of serum was obtained from beneath the raised cuticle of a blistered surface, and gave evidence, when tested, of the presence of bile. The same is found in the yellow fluid of vesicles occurring in the course of the disease. Dr. Blair states that, in a case of recovery from an almost fatal attack of well-marked yellow fever, a great many vesicles or small bullæ appeared over the body and limbs, containing a thin, clear fluid, of a similar yellow colour to that of the skin. The fluid of several of these was collected, and the colour became *green* on the addition of dilute nitric acid. Bile is also found in the serum of blood drawn from the veins or arteries, in the pellicle formed over the crassamentum, as well as in the fluid taken from the heart or large vessels after death.

The analogy, in this respect, between the yellow fever and ordinary jaundice is striking; and if, in the production of the latter, the bile is admitted

¹ Cycloped. of Anat. and Phys., i. 425, 426.

to play an important part, it is difficult to refuse our assent to the opinion which ascribes as important an agency to the same substance in the causation of the yellow discoloration of yellow fever. There is no absorption or regurgitation of bile already formed, for, in by far the largest number of cases, none is secreted. There is merely retention in the blood of those elementary or constituent parts of that fluid which ought to have been eliminated from the system, but of which, from its peculiar morbid condition, the liver has not been able to rid the system. This view reconciles, to a certain extent, both doctrines; for, though the jaundice is doubtless due, not to bile, properly so called, but to a peculiar condition of the blood, the latter owes the power of discolouring the tissues to biliary elements; and if that discoloration differs in some respects from that of ordinary jaundice, the difference, in all probability, depends, as long ago surmised by Warren, on the attenuated, dark, and diseased condition of the blood, and on the sluggish manner in which it circulates in the cuticular capillaries. Indeed, there can be no doubt that the bronze and mahogany hue, the mottled and other unusual appearances of the skin described in the preceding pages, are due to a sanguine exhalation, or to a mixture of tints—of the yellowness arising from the jaundice proper on the one hand, and, on the other, of the dark or violet red of the skin produced by the altered and almost stagnated blood contained in the capillaries of the skin. The correctness of this opinion is shown by the fact that, if a part so coloured is pressed within the finger, the bronzed or mahogany hue disappears more or less completely; but the yellow colour remains unchanged, and as the blood returns in the capillaries, the dark colour in question returns with it, and the skin resumes the peculiar appearance it had before.

It can scarcely be necessary to dwell on sundry fanciful views advocated relative to the cause and nature of the symptom in question. No one can now believe that it is due to the direct agency of the pestilential miasm on the external surface, as taught in former days by a distinguished medical philosopher of our country—the late Dr. Mitchell, of New York; or simply to an increased secretion of sebaceous matter in the skin, as suggested by Fordyce (p. 74); or, again, to the effect of putrefaction, as believed by Curtin;¹ to a morbid and general deposit of dark pigment, as discovered by Hensinger; to “the blow which, in affecting morbidly (*frappant*) the vital principle, destroys the equilibrium, from which results the derangement of the functions of which it is the regulator and the director;”² or to the oxidation of the water exhaled from the blood and impelled to the periphery of the body by the centrifugal force originating in the vital action, and there arrested in the reticular tissue of Malpighi!³

¹ Med. Com., ix. 238.

² Dalmas, p. 13.

³ Magne, Ann. Mar., 1844, iv. 562.

CHAPTER X.

STATE OF THE SKIN CONTINUED.

DISEASES OF THE SKIN.—The skin becomes, in some cases of yellow fever, the seat of morbid changes, besides those already mentioned, which, though not peculiar to, or pathognomonic of, the disease, and though not of constant or frequent occurrence, are nevertheless sufficiently often encountered, and of a sufficiently marked character—to say nothing of their importance as instruments of critical change, and as affording means of arriving at a correct prognosis—to deserve a notice in this place.

1. *Petechiæ*.—This eruption, or one somewhat akin to it, was, as we learn from Dr. Rush, observed during several of our early epidemics (iii. 69, 212). In the fever of 1793, petechiæ were common in the latter stage of the attack. They sometimes came on in large, and at other times in small red blotches; but they soon acquired a dark colour, and, in most cases, were the harbingers of death. Besides these, many persons had eruptions which resembled mosquito-bites. They were red and circumscribed, appearing chiefly on the arms, but sometimes extending to the breast. The former are mentioned as having been of occasional occurrence on the same occasion by Deveze, and the latter appears to have attracted the notice of Nassy. “In the decline of the second stage,” says Cathrall, in speaking of the same epidemic, “the skin was sometimes covered with petechial eruptions, appearing like the stings of nettles of a dark red colour, principally about the neck and breast.” The symptom more particularly belonged to the third or last stage, when the spots increased and became of a purplish colour—the skin between them being of a deep yellow. The same kind of eruption is mentioned by Pascalis, in his account of the fever of 1797 (p. 34); by Ffirth, in his history of that of 1802 (p. 28); and has occurred in some cases in all subsequent visitations.

As may readily be presumed from the results obtained in the yellow fever of this city, petechiæ, or something of analogous nature, have been noticed more or less frequently in other sections of this country,¹ as well as in Europe²

¹ Moultrie, p. 5; A. Hosack, p. 17; Seaman, p. 10; E. H. Smith, p. 126; Monson, p. 181; Pascalis, Med. J. of Marseilles, v. 142; Harrison, N. O. J., ii. 135; Girardin, p. 56; Baxter, Med. Repos., xxi. 4; Archer, Record., v. 68; Stone, N. O. J., ii. 558; Waring, p. 46; Townsend, p. 153; Irvine, p. 29; Lewis, N. O. J., i. 300.

² Arejula, p. 173; Palloni, p. 6; Blin, p. 8; O'Halloran, p. 127; Rochoux, p. 460; Pym, p. 234; Pariset, p. 433; Amiel, in Johnson, p. 267; Smith, Edinb. J., xxxv. 42; Salamanca, p. 17; Tenero, p. 20; Boyd, in Johnson, p. 301; Proudfoot, Dublin Hosp. Repts., ii. 260; Flores, p. 42.

and various parts of tropical regions.¹ But though, from the experience obtained in this city and elsewhere, the occurrence of the eruption in question is placed beyond the possibility of doubt, it is equally certain that it is far from showing itself as frequently in the yellow fever as it does in certain forms of kindred diseases prevailing in northern climates, or the cold seasons of middle latitudes, and particularly in that form of typhoid disease to which it has given a name. In the yellow fever, petechial spots constitute, at best, but an epiphenomenon, occurring generally only in the smaller number of cases; varying in point of frequency in different epidemics, and at times failing completely. Thus, Arejula informs us that he saw none in 1800 at Cadiz; that at Medina Sidonia, in 1801, and at Malaga in 1803, he saw a great many; while in Andalusia, in 1804, they held a middle course, in point of frequency, between the afore-mentioned cities at the periods in question (p. 143). Gillkrest (p. 272) states that petechiæ, "mentioned by a few authors," did not present themselves under his observation in Gibraltar, in 1828. And Dr. Musgrave² remarks that, at Antigua, in 1817, he never met with or heard of them.

Be this as it may, when petechial spots occur, they do so principally in the worst and malignant grades, and in the closing stage of the disease. They are observed as well on the mucous membranes as on the external surface of the body. Those occurring in the former membrane will be made the object of special consideration when we reach the subject of the pathological anatomy of the disease. The spots which show themselves on the external surface are, at times, very numerous, covering the chest and trunk generally, as also the arms and thighs. In other instances, they are sparsely dispersed. They are of an obscure red, violet, and livid colour, irregular in point both of size and shape, without elevation, and, in the few cases that recover, do not give evidence of desquamation.

In regard to the mode of formation and to the nature of these spots, some difference of opinion has existed; for while by some they are regarded as partaking of the nature of true petechiæ, constituting a true cutaneous eruption, with local morbid change in some parts of the substance of the skin, others regard them as the result of a simple effusion of blood in or under that membrane. On this subject I shall speak in a future chapter.

2. *Livid Spots and Vibices*.—In some cases, the petechial spots are replaced by, or associated with, large, livid, ecchymotic spots; and, in some instances, distinct and well-marked vibices make their appearance. In regard to the latter, Dr. Rush states that Dr. John Duffield, who acted as house-surgeon and apothecary at the City Hospital in 1797, informed him that he

¹ Rouppe, p. 306 (Tr., p. 410); Warren, p. 15; Hillary, p. 152; Moseley, p. 488; J. Clark, p. 12; Desportes, i. 194; Chisholm, i. 158, 159; Gillespie, p. 42; Caillot, p. 19; Bally, p. 246; Savarésy, p. 277; Bancroft, p. 13; Maher, p. 849; Rufz, p. 15; Rochoux, pp. 269, 337; Kéraudren, Arch. Gén., xv. 459; Fournier, Dict. des Sci. Méd., xv. 339; Moreau de Jonnes, p. 249; Jolivet, p. 11; Vincent, p. 24; Heastie, p. 21; Anderson, p. 8; Copland, iii. 139; Joubert, p. 967; Hume, p. 209.

² Med.-Chir. Trans., ix. 133.

saw them in many cases, and they were all more or less sore to the touch (iv. 12). Dr. Ffirth also observed them in the epidemic of 1802 (p. 28), and they have been seen at other times. Labat, in one of the earliest accounts we have of the West Indian yellow fever, notices the existence of purple blotches.¹ They are mentioned by Chirac as occurring in the fever of Rocheford in 1694.² They are also, as well as vibices, spoken of by numerous writers on the fever of temperate and tropical climates. Indeed, they are, ecchymotic blotches particularly, noticed everywhere and in very many cases.³

Though occurring on all parts of the surface, these blotches particularly affect the neck, chest, shoulders, and back, as also the eyelids and elbows; and, indeed, all other parts of the body which suffer compression from any cause. They vary in size, being sometimes only a few lines, at others six or eight inches in diameter. They vary also in respect to shape and number, being sometimes but sparsely distributed, in other instances greatly multiplied over the parts mentioned. In point of colour they are purple or violet, sometimes black. Superadded, as they are generally, to a jaundiced discoloration of a more or less deep shade, or presenting at their margins a yellow or greenish hue, they impart to the skin a peculiar aspect, which it is somewhat difficult to describe accurately, but some idea of which may be formed by comparing it with a familiar object which it closely resembles. "When," says Dr. Wilson, "yellow leather gloves are worn on horseback, in rainy weather, and the glove of the bridle hand is thoroughly wet, the palm part of it exhibits a very just likeness of what we see in the cases in question—turbid white, and livid or black, intermixed with imperfect margins of yellow or green, or a hue in which yellow and green are blended; and it is difficult to say which predominates."⁴

With the exception of those affecting the eyelids and elbows, and appearing in the first—though generally at the commencement of the second—stage, ecchymotic blotches seldom if ever show themselves but at an advanced period of the disease. They have usually been viewed as indicative of the most imminent danger, not only owing to the morbid change they indicate in both the solids and fluids of the body, but because they seldom fail to be accompanied with other symptoms of a dangerous import. The only exception to this statement relative to the significance of these blotches in a prognostic point of view, is in regard to those that occur on the eyelids and elbows, which are not, as frequently as the others, the precursors of a fatal issue.⁵

They are the legitimate offspring of the remarkably languid capillary circulation, which serves, in some measure, to characterize the disease, and

¹ *Nouveaux Voy. en Amérique*, ii. 3, iv. 308.

² *Traité des F. Malignes*, i. 53.

³ Rochoux, pp. 117, 337, 461, 497; Monson, p. 181; Alex. Hosack, p. 17; Moultrie, p. 5; Waring, p. 46; Bally, p. 284; Kelly, xiv. 379; Pariset, p. 432; Amiel, *Edinb. J.*, xxxv. 267; Boyd, in Johnson, p. 301; Gillespie, p. 42; Rouppe, p. 36; *Ib.*, Transl., p. 410; Moreau de Jonnes, p. 247; Kerandren, *Arch. Gén.*, xv. 247; Proudfoot, *Dublin Hospital Repts.*, ii. 260.

⁴ Wilson on West Indian Fever, p. 21.

⁵ Rochoux, p. 498.

aids in the production of the dark mottled, dusky hue, and mahogany tinge of the complexion noticed in the advanced stage. From this condition of the circulation, dark, partially oxygenated, and altered blood is permitted to accumulate in the capillaries. It moves slowly, the lentor being rendered evident by pressing on the skin, when the part from which the blood had been forced remains pallid some minutes, and only gradually resumes its former colour.

3. *Vesicular Pimples, and Pustular Eruptions*.—Dr. R. Jackson (i. 187) states, that large vesications, resembling the disease termed pemphigus, have appeared in succession on different parts of the body, about the time of crisis, in several instances within his own experience. Something analogous—an eruption of water-blisters—is described by Dr. Rush (iii. 69). These, after bursting, ended in deep black sores. An eruption of kindred nature is mentioned by Dr. Pascalis, of New York,¹ and Dr. Drysdale, of Baltimore.² Similar observations were made at Barcelona³ and Cadiz.⁴ Phlyctenæ and pimples, on different parts of the body, are mentioned by writers on the yellow fever of tropical and temperate regions.⁵ I have several times seen a crop of hardish pimples over the chest and abdomen. Imray makes mention of a pustular eruption, not unlike the smallpox, and extending all over the body.⁶ Long before his time, Rouppe had remarked that “those who stood their ground against this disorder, and lived to the fifth or seventh day, had small red pimples all over their body, which were very painful, and suppurated with great difficulty. Their whole body was covered with them in a manner very like the confluent smallpox.”⁷ The same variculous kind of eruption was observed by Frost, at Demerara, in 1803 and 1804.⁸ It was noticed in New York in 1822 by Pascalis,⁹ by Gilbert, at St. Domingo, in 1802 (p. 78), Chisholm, at Grenada, in 1793 (i. 179), Blicke at Jamaica.¹⁰ “There sometimes happens,” says Blane, “an eruption of some pustules, with white heads on the trunks of the body” (p. 439).

4. *Scarlatinous, Erysipelatous Inflammation, &c.*—Dr. Rush met, in 1793, with cases of an eruption resembling that which occurs in the scarlet fever (iii. 68). A similar phenomenon, as well as erysipelatous inflammation, was observed on the face, trunk, and extremities, during the epidemic of Barcelona in 1828.¹¹ Large erythematous patches were observed occasionally on the knees and elbows during the epidemic of Barbadoes in 1816. They proved very painful, and occurred only in the fatal cases.¹²

¹ Fever of 1819, Marseilles J., v. 142.

² Med. Mus.; i. 131.

³ Rochoux, p. 473.

⁴ Laso, quoted by Rochoux, p. 473.

⁵ Gilbert, p. 78; Hume, p. 224; Palloni, p. 9; Sir G. Blane, Dis. of Seamen, p. 439; Proudfoot, End. Fev. of Spain (Dublin Hospit. Repts.), ii. 260.

⁶ Pariset, p. 434; Fellowes, p. 57; Gillespie, p. 42; Diet. des. Sci. Méd., xv. 339; Drysdale, i. 131; Dalmas, p. 234; Baxter, Med. Repos., xxi. 4.

⁷ De Morbis Navig., p. 307.

⁸ Med. Repos., xiii. 37.

⁹ Marseilles J., v. 143.

¹⁰ Ital. Tr. in Zecchinelli, Feb. Gialla, p. 239.

¹¹ Rochoux, p. 473; Pariset, p. 434.

¹² Ralph, ii. 75.

5. *Eruptions about the Mouth—Pustules—Aphthæ.*—Dr. Rush informs us that, in 1793, there was an eruption about the mouth in many people, which ended in scabs similar to those which take place in the common bilious fever. They always afforded a prospect of a favourable issue of the disease. Such eruptions, which were noticed in our subsequent epidemics, are described as of not unfrequent occurrence in most places where the yellow fever has prevailed, whether in temperate or tropical regions, the assertion of Dr. Gillkrest to the contrary notwithstanding (p. 272).¹

6. *Furuncles and Carbuncles.*—The pustular eruption assumes, sometimes, a severer and more painful character. Arejula mentions (p. 173) the occasional occurrence of carbuncles in the yellow fever of Cadiz, in 1800. The same symptom is mentioned by other writers as occurring in the fever of Spain;² and long before their time it was observed at Rocheford by Chirac (pp. 55, 233, 234, &c.). They, or furuncles, or both, have also been noted by writers on the tropical fever.³ In this country they are spoken of by Davidge, of Baltimore (p. 103); Pascalis (v. 142), Dalmas (p. 234), A. Hosack (p. 19), Townsend (p. 185), of New York; Monson (p. 181), of New Haven; Stone (p. 258), of Natchez; Baxter (*Med. Repos.*, xxi. 4), of New Orleans; Waring (p. 46), of Savannah; *Fever of N. Y. 1798* (*Med. Repos.*, ii. 199); Lewis (*N. O. J.*, i. 300). In this city, carbuncles have been often seen. They are specially mentioned by Dr. Monges⁴ and Dr. Caldwell.⁵ "Several cases," says Dr. Rush, "such as occur in the plague, came under my notice. They were large and hard swellings on the limbs, with a black apex, which, upon being opened, discharged a thin, dark-coloured, bloody matter. From one of these malignant sores a hemorrhage took place" (iii. 69). In New Orleans, in 1853, furuncles were very common over the whole body. They occurred during convalescence, and even in many who had not the fever.⁶ They were common in the same city in 1848.⁷

7. *Miliary Eruptions.*—Nor is it uncommon to see cases attended with a full crop of miliary pimples. They have been noticed in this country, in Europe, and in the West Indies.⁸ The prickly-heat and the nettle-rash have also been observed.⁹ Dr. Barton, in his report on the sanitary condition of New Orleans in 1853, says that, during the epidemic of that year, "a fine

¹ Towne, p. 51; Arnold, p. 19; R. Jackson, pp. 63, 76, 187; Seaman, p. 10; Townsend, p. 153; Kelly, p. 379.

² Fellowes, p. 56; Blin, p. 22; Rochoux, p. 473; Flores, p. 42.

³ Desportes, i. 197, 198; Warren, p. 19; Rouppe, p. 307; Gilbert, pp. 77, 78; Pugno, p. 360; Rochoux, p. 346; Blane, p. 439; Chisholm, i. 177; Fontana, p. 74; Lind, p. 285; Gillespie, pp. 41, 42; Lefort, p. 561; Hunter, p. 77; Frost, *Med. Repos.*, xiii. 37; Davidson, *Med. Repos.*, i. 165; Kerandren, *Arch.*, xv. 459; Lallemand, *Fev. of Rio*, p. 118.

⁴ *N. O. J.*, ii. 62.

⁵ *Analogy of Y. F. and Plague*, p. 19; *Ib.*, *Fev. of 1803*, *Med. Repos.*, vii. 180.

⁶ Barton, *Rept. on Sanitary Condition of N. O.*, p. 245.

⁷ *N. O. J.*, vi. 37.

⁸ Pariset, p. 434; Fellowes, p. 57; Gillespie, p. 42; *Dict. des Sci. Méd.*, xv. 339; Drysdale, i. 131; Dalmas, p. 234.

⁹ Gillespie, p. 42.

miliary eruption was usually seen on the skin within twenty-four hours from the attack. It was the harbinger of safety as long as it kept out; its repulsion the signal of great danger, if not of fatality" (pp. 244-5). In his account of the fever of Antigua, in 1853, Dr. Furlonge states: "In a young Scotchman, who nearly fell a victim to the fever, I observed an eruption on his arms, body, and legs, resembling measles, exactly such as is described by Dr. Graves in his *Clinical Medicine* as remarked in maculated typhus. I requested Dr. Robertson, of the army, the P. M. officer here, to see him with me, as I had never seen such an eruption in the yellow fever or typhus; he told me it was like the exanthema he had observed in Ireland in typhus."¹

8. *Phlegmonous abscesses*, even, have occasionally been noticed in various parts of the body, and at times give rise to considerable inconvenience and suffering. When once discharged, they seldom fill again. Arising, sometimes, after convalescence from the last stage of the disease, they often retard the restoration to perfect health, and have occasionally proved fatal. In general, however, the incipient formation of an abscess—other symptoms being favourable—may be viewed as the harbinger of a happy issue.²

9. *Anthrax*.—This morbid condition of the skin has now and then been observed to form on some part of the body. It usually appears in the closing stage of the disease, and is generally, though not always, a sign of unfavourable omen. In New York, in 1822, out of 425 cases, there were only two instances of anthrax.³ In 1793, they were not noticed by Deveze, (pp. 32-46), or Nassy (p. 22); but Dr. Rush (iii. 69) mentions a case which occurred that year, and "succeeded a favourable issue of the disease." Cases of the kind are mentioned by Caillot (p. 22), Gilbert (p. 73), Desportes (i. 129-197-199-214-223), Chisholm (i. 179), Savarésy (p. 277), Kelly (p. 379), Pasealis (*Marseilles Journ.*, v. 142), Arejula (p. 173), Fellowes (p. 56), Davidson (*Med. Repos.*, i. 166), *Report to Med. Soc. of N. Y. on Epidemic of 1798*, p. 7, (*Med. Repos.*, ii. 214), Dalmas (p. 9), Kéraudren (*Arch.*, xv. 459), Chiras (i. 55-230-234), &c.

10. *Gangrene of the Skin*.—Dr. Rush remarks, in reference to the epidemic of 1793, that, notwithstanding the disposition to cutaneous eruptions which existed in the disease of that year, it was remarkable that blisters were much less disposed to mortify than in the common nervous fever. He met but one case in which a deep-seated ulcer followed an application of the kind to the legs (iii. 69). Dr. Townsend saw nothing of the kind in New York in 1822 (p. 165). Neither was such an effect noticed in Gibraltar during the epidemic of 1828, by Dr. Gillkrest, who, relying on the result of his personal experience, would seem disposed to entertain doubts as to the reality of the occurrence on other occasions.⁴ By others, however, the gangrene of blistered surfaces appears to have been more or less frequently en-

¹ Lancet, 1853, ii. 441-2.

² Rochoux, p. 522; Pariset, p. 434; Cathrall, p. 39; Blin, p. 22; Bally, p. 253; Hunter, p. 82; Sir J. Fellowes, p. 57; Drysdale, i. 131; Gilbert, p. 78; R. Jackson, i. 188; Blair, p. 68.

³ Townsend, p. 153.

⁴ Cyclopedia, ii. 272.

countered. It was common at the Bush Hill Hospital in 1793, according to Deveze (pp. 32, 37, 46, 92), and is noticed by other writers. So also the gangrene of excoriated parts—of the sacrum, the prepuce, and scrotum in men, and the vulva in females; of the rectum, anus, trochanter, and lower extremities. Such phenomena have all been occasionally observed in the last stage of the fever of both temperate and tropical regions.¹

11. *Parotitis and Buboes*.—Besides the phenomena mentioned, as manifesting themselves in the skin, others present themselves occasionally, which, though seated primarily in a different order of parts, and affecting the cutaneous covering in a secondary way, and only to a limited extent, will be, for the sake of convenience, mentioned in this place. I allude to glandular swellings of various parts of the body, though particularly of the parotid, inguinal, and axillary glands.

Parotitis, though far from being of as common occurrence in this fever as in the oriental plague, and though in fact seldom met with, has been noticed sufficiently often in this country, in Europe, and in tropical regions, to be regarded by some as constituting a link between the two diseases. They are mentioned by the historians of the epidemics of this city—by Rush (iii. 67–211), Deveze (pp. 32–39), Cathrall (p. 39), and are referred to in the numerous accounts we possess of the disease as it has appeared elsewhere.²

Whether, as Bancroft (p. 50) and Savarésy (p. 291) remark, the swelling of the parotid glands, which, according to their own showing, is sometimes seen in the yellow fever, differs greatly from the glandular tumours which occur in the plague, and in what the difference, if real, consists, I am not prepared to say, or disposed to inquire. But be this as it may, such tumours appear late in the disease. They sometimes suppurate. At others, after exhibiting more or less inflammation, they disappear without the formation of purulent matter, and may generally, though not always, be regarded as a favourable omen.

Buboes of the groin and axilla are probably of rarer occurrence in the yellow fever than the affection of the parotid glands—of so unusual an occurrence, indeed, that, by some writers,³ a line of demarcation between that disease and the oriental plague has been established, predicated on the absence of this symptom in the former and its constant attendance on the latter.

¹ Moultrie, p. 22; Chisholm, i. 169; R. Jackson, i. 188; Lefort, De la Saignée, p. 561; Bally, pp. 250, 1; Savarésy, p. 277; Pascalis, Journ. of Marseilles, v. 142; Girardin, p. 56; Rufz, p. 15; Kelly, xiv. 379; Pariset, p. 434; Rochoux, pp. 461–474; Maher, p. 854; Perez, Gén. de Méd., lxxviii.; Kéraudren, Archiv. Gén., xv. 459; Desportes, i. 129; Blair, p. 65; Arejula, p. 173; Flores, p. 52, note; Lallemant, p. 119.

² Drysdale, i. 138; Monson, p. 181; Waring, p. 46; Williams, p. 35; Desportes, i. 199; Ralph, ii. 77; Gilbert, p. 73; Madrid, pt. ii. p. 30; Savarésy, p. 277; Catel, Ann. Mar. iv. 1844, p. 227; Pugnet, p. 360; Lefort, p. 561; Gillespie, p. 43; Arnold, p. 10; Lind, p. 285; Bancroft, p. 50; Anderson, p. 11; Rochoux, pp. 346–524; Blicks, p. 205; Maher, p. 849; Hunter, p. 81; Catel, Rept., p. 16; Boyd (in Johnson), p. 300; Arejula, p. 173; Chirac, i. 55–230, &c.; Fournier, xv. 338, 9; Pariset, pp. 341–433; Chisholm, i. 178; Vatable, p. 346; Frost, xiii. 36; Blin, p. 22; Velasquez, p. 14; Pym, pp. 60–63; Fellowes, p. 57; Fev. of Cayenne, 1850, p. 178; De Maria, p. 73; Lallemant, p. 118.

³ Blin, p. 38.

Whatever may be the value of the difference in question in a diagnostical point of view, the history of the disease will show that the absence of buboes is not constant in the yellow fever, and that so far from this, they have been noticed in the epidemics of this and other cities. Though escaping completely the observation of Deveze (ii. 32-46) and Nassy, in the fever of 1793, they were seen in three instances by Dr. Rush. "They were extremely painful in one case in which no redness or inflammation appeared. In the others, there was considerable inflammation, and but little pain. All these patients recovered without suppuration of their swellings. In one case, the whole force of the disease seemed to be collected in the lymphatic system. The patient walked about, and had no fever nor pain in any part of his body, except in his groin." In another case which came under Dr. Rush's care, a swelling and pain extended from the groin along the spermatic cord into one of the testicles (iii. 67, 8). The same writer met with two cases in 1794 (iii. 211). Buboes are also mentioned by Dr. Monges¹ and Dr. Caldwell,² of this city.

These glandular swellings have been noticed by Chirac at Rochefort in 1694 (i. 55, 230-234). In the same year, at Martinique, by Labat;³ in 1796, by Davidson;⁴ and in 1802 by Savarésy (p. 277) and Moreau de Jonnés (pp. 105-298), in the same island; Bally (p. 250) observed them at St. Domingo in 1801, and it will be easy to collect references to many other writers who have met with them in this and other countries.⁵

Buboes, like other symptoms observed in the yellow fever, vary in point of frequency in different places, and at different returns of the disease in the same place; being totally absent in some localities or seasons, and occasionally, or even frequently, met with in others. They, as well as parotitis, were comparatively often encountered at Martinique in 1802;⁶ they were seldom observed at Cadiz in 1800—never, according to Blin;⁷ and never, also, as it would appear, in New York in 1795,⁸ and Barcelona in 1821.⁹ Dr. Rush says of the fever of this city in 1797: "Glandular swellings were very uncommon in this fever. I should have ascribed their absence to the copious use of depleting remedies in my practice, had I not been informed that morbid affections of the lymphatic glands were unknown in the City Hospital, where bloodletting was seldom used" (iv. 12).

Like the swelling of the parotids, the tumours in question sometimes suppurate; at other times, they disappear with the progress of convalescence, and furnish the same prognostical indications as the former.

¹ N. A. Med. and Surg. Journ., ii. 62.

² Med. Repos., vii. 180; Essay on Plague, p. 19.

³ Nouv. Voy. en Amérique, i. 72, 3.

⁴ Med. Repos., i. 165.

⁵ Kelly, p. 379; Report to Med. Soc. of N. Y. on Epid. of 1798, p. 7; Med. Repos., ii. 214; Williams, Bil. Fev. of Jamaica, p. 37 (1750); Lind, p. 285; Warren, p. 18; Arnold, p. 10; Fournier, Dict. des Sci. Méd., xv. 338; De Maria, p. 73; Pym, pp. 60-63; Girardin, p. 56; Chisholm, i. 177; Blicke, p. 205; Monson, p. 181; Fever of Cayenne (1850), p. 178; Proudfoot, Dub. Hosp. Rep., ii. 263.

⁶ Dict. des Sci. Méd., xv. 338, 9.

⁷ Blin, p. 22.

⁸ A. Hosack, p. 18.

⁹ Rochoux, p. 522; Pariset, p. 433.

CHAPTER XI.

DIGESTIVE ORGANS.

STOMACH.—In the general description of the disease, it was stated that, in almost every case, the gastric organs become early involved in the morbid derangement; that nausea, with or without vomiting, is a rather frequent attendant from the outset of the attack, and that the stomach is often distended; sometimes—though not always—painful on pressure; and irritable after the ingestion of drink or food, with a greater or less propensity to eject its contents, especially when pressure is applied to the epigastric region. In addition, it was stated that, although these symptoms often occur at an early period, they as often appear later, and are not generally fully developed until from twelve to twenty-four hours from the outbreak of the attack—sometimes not before the accession of the second stage, when they acquire prominence and become highly distressing, and are attended with burning heat at the præcordia, and acute, sometimes excessive tenderness on pressure. It was also mentioned that, as the disease advances, the phenomena in question become aggravated, and continue generally to harass the patient till near the approach of death, when they sometimes diminish in violence and even subside altogether; that the matter vomited, which at first consisted merely of the substances swallowed, combined with glairy mucus of an acid taste, and sometimes, though not often, except in mild cases, mixed with bile, becomes streaked of a red or brownish colour; and that it consists of brown, blackish or chocolate-coloured flakes or particles diffused in a colourless liquid; which, though at first only slightly tinged with them, ultimately becomes black and opaque, and acquires the appearance of coffee-grounds floating in a serous fluid. It was, moreover, stated that, in some instances, grumous, dissolved or pure blood is thrown up, the matter ejected being sometimes so acrid as to excoriate the throat, tongue, and lips; and at other times, of insipid smell or taste; that the quantity of fluid thus ejected is generally very great—sometimes extraordinarily large—the emptying of the stomach being effected by a sudden contraction of that organ and other parts concerned, which propels the fluid ejected to a considerable distance; and, finally, that this black matter, though met with generally in bad or fatal cases, fails often to appear during life, and is not, necessarily, followed by death.

These statements of the morbid affection of the stomach in the several stages of the yellow fever, as it presents itself in this city, are founded on the results of my own observations, and rest besides on the testimony of Rush (iii. 59, 205–220), Currie (pp. 21, 22–24, and *ib.* on *Bil. Fev.*, p. 219),

Deveze (pp. 21–24), Nassy (p. 20), Barnwell (pp. 369–372), S. Jackson (p. 59), Cathrall,¹ Caldwell,² Ffirth,³ and Pascalis.⁴

They accord in every particular with the phenomena observed in other places visited by the disease, whether in this country, in the South of Europe, or in tropical climates.

1. If, with us, the early manifestation of nausea and vomiting is noted, it has not the less been found to constitute a most prominent feature in the disease from Boston to Texas.⁵ In Leghorn, Gibraltar, and the various cities of Spain;⁶ also in all the West India Islands, and on the coasts of Africa and of Southern America;⁷ the period of accession or full development of these symptoms varies as with us, from the first onset of the attack, to twelve or twenty-four hours after, or, in some cases, to the subsidence of the stage of febrile excitement.

2. If, in the fever of this city, the matter vomited consists at first merely of the substances swallowed mixed with a glossy mucus, more or less ropy and viscid, and acid to the taste—and sometimes, though not often, except in mild cases, with bile in any quantity—colourless, or light-coloured, yellow, or green; so in other parts of these States, the matter ejected in the first stage of the fever has, in most cases, been found to present like characters, and especially to exhibit generally, except in mild cases, and under peculiar circumstances of idiosyncrasy, of complication and critical changes, a similar deficiency of bilious matter.⁸ Not different have been the phenomena observed

¹ Philosophical Trans., v. 118; *Ib.*, Sketch of Fever of 1793, p. 24, &c.

² Fever of 1805, pp. 81–5.

³ A Treatise on Malignant Fever, &c., p. 25, &c.

⁴ Fever of 1797, p. 30, &c.

⁵ Lining, ii. 413; Moultrie, p. 3; S. Brown, p. 12; Irvine, p. 30; Valentin, p. 166; Dalmas, p. 6; Gros, p. 9; Cartwright, ix. 13; Drysdale, i. 154; Archer, v. 66; Tully, p. 295; Ashbel Smith, *Am. Journ.*, xxv. 501; Warren, p. 501; Seaman, p. 9; E. H. Smith, p. 119; Monson, pp. 179, 180; Ticknor, iii. 222–4; C. Drake, xxi. 134; Baxter, xxi. 3; Barton, xv. 37; Dickson, iii. 253; Merrill, ii. 222; *N. O.*, 1820, p. 8; *N. O.*, 1839, p. 331; Barton, *Fev. of 1833*, p. 10; Shecut, p. 119; Davidge, p. 103; Randolph, *Med. Repos.*, xxiii. 168; Addoms, *Fev. of N. Y. in 1791*, p. 9.

⁶ Palloni, p. 4; Dufour, iv. 51; Caisergues, p. 166; Berthe, pp. 82–4; Blin, p. 6; Velasquez, p. 10; Pariset, *Obs. &c.*, p. 29; Arejula, p. 159; Doughty, p. 126; Pym, pp. 228–9, Pariset, p. 388; O'Halloran, p. 79; Audouard, p. 57; Louis, p. 217; Rochoux, p. 477; R. Jackson, *Fev. of Spain*, p. 71; Jourdain, v. 257; Flores, p. 39.

⁷ Warren, p. 10; Towne, p. 21; Chisholm, i. 149; Lempriere, ii. 83; Ralph, ii. 65–67–70; McArthur, p. 346; Savarésy, pp. 271, 2; Hillary, p. 147; Gillespie, p. 38; Desportes, i. 193; Pugnet, p. 355; R. Jackson, *Sketch*, p. 108; Moseley, p. 436; Th. Clark, p. 10; Imray, liii. 79; Bancroft, p. 30; Osgood, p. 10; Barrington, xii. 311; Peixotto, i. 413; Frost, xiii. 298; Dubreuil, *Journ. Univ.* viii. 321; Caillot, p. 16; Barry (in Boyle), p. 271; Rochoux, p. 276; Mabit, p. 10; Jolivet, p. 8; Bourdon, p. 9; Cayenne, 1850, pp. 167–172; Hume, p. 197; Heastie, p. 18; Copland, iii. 139; Carter, p. 4; Anderson, p. 4.

⁸ Hosack, *Practice*, p. 319; Warren (in Tytler), p. 501; Lining, ii. 413; Drysdale, i. 134; Kelly, xiv. 378; Gros, p. 9; Cartwright, ix. 11; Girardin, p. 33; Ticknor, iii. 222; Townsend, p. 154; Archer, v. 66; Irvine, p. 30; Tully, p. 296; Valentin, p. 166; Smith (Texas), xxv. 502; Seaman, p. 9; *N. O.*, 1820, p. 8; Waring, p. 5; E. H. Smith, p. 120;

in Europe,¹ where the dejections have been found to be, most generally, of the kind mentioned, and where, if the matter thrown up is tinged with bile, more or less pure, and in greater or less quantity, the mixture has as often been found to fail, and cannot hence be regarded otherwise when it occurs, than as an accidental complication. The same results have been obtained elsewhere, and are recorded in all the writings of tropical physicians from the days of Towne to our own.²

In a word, the fever, as well in regard to this as to other symptoms, has everywhere, and at all times, presented the same phenomena—the matter vomited in the early part of the attack, and during the first stage, consisting, at first, of the contents of the stomach, often of undigested food, and of a glairy, ropy, viscid mucus of an acid taste. Sometimes, though not always, a greater or less quantity of bile, proceeding from the gall bladder compressed in the act of emesis, is thrown up. As the disease progresses, the bilious matter, if at the outset vomited, ceases, in uncomplicated cases, to show itself, whatever be the place where, or the time at which the disease is observed. The substance vomited continues, up to the appearance of the black vomit, to consist, besides the drinks or medicines taken, of a mucous fluid of increased acidity, and often of a bluish tinge, and at last assumes, in many cases, a peculiarity to which attention will be called in the next chapter. The identity of the fever, in all latitudes, on the point in question, is complete. It may be remarked, that the substance to which the acid taste of the matter vomited is owing, is free hydrochloric acid, to the excessive elimination of which, even at the outset, there is a great tendency in the disease, and which, as will be seen, plays an all important part in the formation of the black vomit. Dr. Wragg, of Charleston,³ well remarks, that to the presence of this acid in such large quantities is doubtless owing the blue colour which the vomited fluid often presents, as also the undigested state of the last meal eaten by the patient previous to his attack, which is generally thrown up pretty much as it has been swallowed, even many hours after it has been in the stomach.

Monson, p. 179; Bayley, p. 83; Stone, N. O. J., vi. 553; Davidge, p. 103; Harrison, N. O. J., ii. 132; Thomas, p. 83; Wragg, *op. cit.*, p. 76.

¹ Berthe, p. 82; Fellowes, p. 55; Caisergues, pp. 166, 7; Amiel (in Johnson), p. 263; Blin, p. 6; Velasquez, p. 11; Rodriguez (in Pariset), p. 29; Gillkrest, ii. 271; Palloni, p. 4; Edin. Journ., ii. 84; Proudfoot, xxvii. 250; Audouard, pp. 56, 7; Pariset, pp. 372, 3–289, &c., 420; Arejula, p. 159; O'Halloran, p. 80; Doughty, p. 126; Pym, pp. 60–2; Rochoux, p. 477; Robt. Jackson, *Fev. of Spain*, p. 96; Flores, pp. 39, 40.

² Towne, p. 21; Bruce, p. 278; Chisholm, i. 174; Lempriere, ii. 64; Savarésy, p. 273; Moseley, pp. 436, 7; McArthur, p. 347; Bancroft, p. 31; Osgood, p. 10; Madrid, p. 25; Blanc, p. 405; Gillespie, p. 38; Ralph, ii. 73; Pugnet, p. 355; Vatable, p. 345; Evans, p. 233; Dickinson, pp. 128–134; Arnold, p. 9; Bally, p. 218; Hillary, p. 147; Barrington, xii. 311; Maher, p. 842; Caillot, p. 18; Belcher, p. 250; Dariste, p. 160; Gilbert, p. 66; Wilson, p. 182; J. Clark, p. 7; Rochoux, pp. 276, 7; Hunter, 64–67; *Diet. des Sci. Méd.*, xv. 336; R. Jackson, *Sketch, &c.*, i. 105–108–110; Vincent, p. 23; Jolivet, p. 8; Heastie, p. 18.

³ Charleston J., x. 76–7.

3. If, in the fever of this city, the vomiting, whether coming on early or one or more days after the commencement of the attack, has, with few exceptions, proved a prominent and distressing symptom—continuing, in fatal cases, to harass the patient till the approach of death, or a short time previous, becoming aggravated as the disease advances, and being, in many instances, irrepressible, and excited by pressure on the epigastrium—the yellow fever of other places, both on this and the other side of the Atlantic, in tropical and extra-tropical regions, has generally been characterized by like phenomena. In all our infected cities, vomiting is found to be a frequent attendant—to be easily excited by pressure or otherwise, and to prove distressing to the very close of the attack—sometimes to within a few hours of death.¹ In Europe, excessive irritability of the stomach, frequent—almost constant and irrepressible—vomiting is noted by every writer as a usual attendant on the disease, constituting one of the principal sources of the distress and suffering experienced by the patient.² “As the disease proceeds in its course,” Dr. Gillkrest remarks, in speaking more particularly of the fever of Gibraltar, “the irritability of the stomach usually becomes one of the most remarkable as well as indomitable of the symptoms; there is often, indeed, little use in directing medicines or drinks, even of the most delicate kinds, and in the smallest quantities, as all are instantly rejected; and, altogether without resources, we often find ourselves obliged to look on in the expectation of the arrival of a tranquil moment, when we may again venture on the exhibition of something.”³

While such is the case in this country and Europe, it is only necessary to glance at the descriptions we have of tropical yellow fever, to discover that the frequent and intractable nausea and vomiting, excited by the ingestion of solid or liquid substances, by merely moving in bed or sitting up, or by pressure, constitute a main feature of the disease, continuing to the last, or ceasing some hours before death, as from paralysis of the stomach.⁴

¹ Lining, ii. 413, 419; Dalmas, p. 6; Gros, p. 10; Cartwright, ix. 11; Drysdale, i. 134; Merrill, ii. 222, ix. 244; Dickson, iii. 253–5; Ticknor, iii. 222, 5, 7; Townsend, pp. 154, 161, 3; Hill, v. 90; Irvine, p. 28; Repert., x. 516; Tully, p. 295; Valentin, p. 166; Baxter, xxi. 3; C. Drake, xxi. 134; A. Smith, xxv. 501–2; A. Hosack, p. 14; Waring, p. 47; N. O. Fev. 1819, p. 8; Ib. 1820, p. 9; Ib. 1839, p. 332; Seaman, p. 9; E. H. Smith, p. 118; Addoms, p. 9.

² Palloni, p. 4, &c.; Edinb. J., ii. 84–5; Dufour, iv. 51; Fellowes, pp. 53–4; Caisergues, pp. 166–8; Berthe, p. 84; Audouard, pp. 57, 6; Boyd, in Johnson, p. 300; Pariset, p. 30; Smith, xxv. 42; Louis, pp. 217, 226; Gillkrest, ii. 271; Pym, p. 229; Audouard, p. 61.

³ Cyclop., ii. 271.

⁴ Warren, p. 10; Chisholm, i. 149; Pugnet, pp. 353–5; Bancroft, pp. 31–2; Lepriere, ii. 64; McArthur, pp. 347–8; Moseley, pp. 436–8; Imray, liii. 81; Hillary, pp. 147–8; Hunter, pp. 64–67; Madrid, p. 25; Jackson, Tr., p. 261; Ib., Sketch, i. 108; Blane, pp. 410–436; Gillespie, p. 38; Ralph, ii. 66–8; Osgood, p. 10; Barrington, xii. 311, 312; Vatable, p. 345; Peixotto, i. 413; Belcher, xxiii. 250; Dyott, p. 1003; Rufz, p. 56; Rochoux, p. 282; Bally, pp. 218, 239; Lefoulon, p. 68; Catel, An. Marit. 1844, iv. 225; Arnold, p. 9; Holliday, p. 10; Dickinson, p. 132; Caillot, pp. 17, 18; Dariste, p. 162; Dict. des. Sci. Méd., xv. 336.

4. If, after having presented, during a period of longer or shorter duration, and generally until the accession of the second stage, the characters above mentioned, the matter vomited in the yellow fever of this city contains streaks of a red or brownish hue; next consists of brown, blackish, or chocolate-coloured flakes, or particles diffused in a fluid which, at first colourless, becomes ultimately black and opaque, resembling coffee-grounds floating in a serous fluid; if, in some cases, grumous, dissolved, or pure blood, or a substance resembling thin tar or a mixture of soot and water is thrown up; if the matter so ejected has been found, at times at least, to be acrid and exco-riating, and in other instances insipid to the taste and bland in its effect; if the black vomit, though a usual attendant on the severe and fatal forms of the disease, fails to be observed in probably one-third of such cases; if, again, in the fever of this city, the ejection of the black matter is generally preceded or accompanied by a sensation of rawness in the stomach and anxiety and pain at the epigastrium; if this matter is thrown up, often, in very large quantities, and is gulped up or spouted out rather than regularly vomited up; if, finally, the appearance of the black vomit, though usually held as a symptom portending a fatal issue, has not always proved the precursor of death; the yellow fever of other places, in this country and elsewhere, has exhibited similar results, as I shall have occasion to point out in detail in a future chapter.

The irritability of the stomach, and the frequent repetition of the act of vomiting consequent upon it, vary, as to extent, at various periods and in various places. The fever which M. Rochoux observed in the West Indies was characterized by early, considerable, continued, and increasing irritability of that organ, and distressing and frequently repeated vomiting; scarcely one patient in one hundred having escaped, if from the black, at least not from bilious mucous vomiting (p. 276). In the epidemic of Barcelona, in 1822, on the contrary, according to the account given of it by the same writer, those symptoms were much less prominent. At the outset of the disease, it is true, vomiting sometimes came on, and the stomach was quickly emptied of its contents. But this was not usually the case; and, when so relieved, the vomiting soon ceased, or diminished during two or three days, at the expiration of which period it reappeared, and became gradually more frequent as the disease progressed. The black matter was not thrown up in large quantities, and not more frequently than six or eight times in twenty-four hours; while, in rapid cases, many patients died not only without throwing up black matter, but without vomiting at all (pp. 477, 488).

But from this we must not argue, as Dr. Rochoux has done, a difference in the nature of the yellow fever of the two regions; for it would not be difficult to point out an equal difference, at several periods, in the same country. Thus, Tully informs us that, at Middletown, in 1820, "in every case the stomach was *uncommonly* irritable;" and he Italicizes the "*uncommonly*," to show more forcibly the great extent to which the symptom was carried (p. 295). On the other hand, we learn from Dr. Townsend that, in the epidemic which prevailed in New York during the summer and autumn of 1822, the stomach, in

the first stage, was usually calm; the irritability of that organ commenced only during the second, and increased during the third stage (pp. 147, 154, 160). While at Guadaloupe the condition of the stomach was such as described by Roehoux, during the period of his residence there, we find, from Dr. Vatable's account of the epidemic in the same island in 1826, that the stomach was seldom much affected during the first stage (p. 345). Mr. Rufz, in the epidemic of Martinique in 1838, as we learn from Dr. Chervin's able report on his memoir, did not see a single case in which vomiting commenced at an early period and continued without abatement to the end, "as has been observed in other epidemics" (p. 12).

From all this it follows, then, that, in all regions and places where the yellow fever has prevailed—whether it be in this or other cities of the United States, in the sections of Southern Europe which it has visited, or in its more legitimate abode, tropical countries—nausea and vomiting rank among the early symptoms; that they usually, though not always, set in soon after the accession of the disease; that the matter vomited consists at first of the substances contained in or taken into the stomach, mixed with a glairy mucus and other gastric fluids; that, in some cases, bile is at first ejected, the quantity varying in different instances during the same epidemic, according to a variety of circumstances connected with the previous condition of the individual attacked, or the coexistence of other morbid causes, giving rise to a particular complication; that, in general, bile is not thrown up, and, if early ejected, soon ceases altogether to show itself; and that, at the end of a few days, when the disease assumes a malignant character, and manifests a tendency to come to a fatal issue, the matter thrown up assumes the nature of black vomit.

Eructations and Hiccup.—The nausea, irritability of the stomach, and vomiting, which I have just described, but especially those of the early stage, are usually preceded by, or accompanied with, frequent and troublesome eructations.¹ According to M. Roehoux, they are remarkable in this, that they principally occur after drinking, and are, more rarely than nausea, excited simply by motion (p. 282).

While such is the case in the early stage of the fever, the patient is usually troubled in the latter stage with hiccup. This symptom, which often proves distressing, contributing largely to deprive the patient of rest and comfort, varies, like others, as regards frequency and violence, at different periods and in different places; but, whether frequent or otherwise, it has been noticed as an accompaniment of the disease in all climates. In the fever of this city in 1793, it was noticed by Rush, who remarks, however, that it was less frequent than in the last stage of common bilious fever. Other practitioners of that period describe it as a distressing symptom, without hinting at its unfrequency.² But, however this may have been in 1793, we learn from Dr. Rush that, in 1797, hiccup was more frequent and troublesome; and our

¹ Rufz, pp. 12–14; Roehoux, pp. 478–482; Pariset, pp. 373–390; Bally, pp. 218–226; Mabit, p. 10; Anderson, p. 8.

² Currie, p. 27; Nassy, p. 25.

subsequent epidemics have furnished facts to the same effect.¹ Similar results have been noted in other parts of the United States.² At Barcelona, in 1828, it was a frequent attendant on fatal cases, according to Louis (pp. 224, 225), and was noted in the various accounts we have of the other epidemics of that place, as also of those of Spain and of Leghorn.³ At Barcelona, in 1828, hiccup seldom failed to appear in conjunction with black vomit, and constituted one of the most frequent attendants on the last stage of the disease, continuing, without intermission, hours at a time, and recurring after short intervals of respite.⁴ Dr. R. Jackson, who saw the fever in Cadiz and Xeres in 1820, describes hiccup as frequent and troublesome—sometimes before black vomiting had commenced, sometimes after it had been suppressed (p. 102).

Dr. Rochoux, in his account of the yellow fever he observed in the island of Guadaloupe, and which he wrongfully regards as the only type of the West Indian fever, remarks that hiccup was not a frequent attendant, not appearing in more than one case in ten (p. 283). Bally also found it of rare occurrence in the yellow fever of St. Domingo (p. 245). Others, however, at the same time or in different sections of tropical regions, have, as in this country and in Europe, noted its more frequent occurrence.⁵

As a general rule, hiccup may be regarded as a symptom of very unfavourable import, usually indicating a state of great danger, and even approach of death. To this, however, there are, as in respect to other symptoms of like character, exceptions—patients recovering after having suffered from long-continued hiccup.⁶ But while such is sometimes the case, the cessation of that symptom must not invariably be hailed as the harbinger of a favourable change, except perhaps in cases when, together with the occurrence in question, there is an amendment in the other symptoms—for not a few instances present themselves in which the patients, though ceasing to be affected with hiccup, continue to sink under the pressure of the disease.

INTESTINAL CANAL. *Alvine Evacuations.*—In the very large majority of cases of yellow fever, constipation of the bowels constitutes a predominant feature in the early stage, proving, in some instances, as obstinate as in any

¹ S. Jackson, p. 56.

² Lining, ii. 425; Seaman, p. 9; E. H. Smith, p. 117; A. Hosack, p. 16; Valentin, p. 174; Baxter, xxi. 3; A. Smith, Am. Journ. Med. Sci., xxv. 502; Girardin, p. 56; Waring, p. 47; Merrill, ii. 222; Archer, Recorder, v. 67; Tully, p. 299; N. O. in 1839, p. 334; N. O. in 1820, p. 10; Thomas, p. 85; Kelly, xiv. 379; Addoms, p. 11.

³ Pym, Edinb. Med. and Surg. Journ., xxxv. 41; Th. Smith, ib., xxxv. 42; Amiel, ib., xxxv. 266; Ib., in Johnson, p. 264; Dufour, Marseilles Journ., iv. 52; Palloni, p. 4; Gillkrest, Cyclop. Pract. Med., ii. 271; Fellowes, p. 55; Berthe, p. 87; Proudfoot, Edinb. Journ., xxvii. 250; Blin, p. 6; Velasquez, p. 11; Pym on Bulam Fever, p. 234.

⁴ Audouard, p. 63; Rochoux, p. 482; Alf. de Maria, p. 71; Periodico de la Soc. de la Med., No. 3, p. 257; Flores, p. 40.

⁵ Gilbert, p. 66; Osgood, p. 12; Caillot, p. 19; Bancroft, p. 12; Dyott, p. 1003; Gillespie, p. 4; J. Clark, p. 14; Pugnet, p. 357; Ralph, ii. 67-73; Vatable, p. 346; Imray, liii. 81; Savarésy, pp. 273-277; Dict. des Sci. Méd., xv. 337; Dickinson, pp. 126-131.

⁶ Rush, iii. 64; Jackson, p. 56.

known disease, and resisting at times the action of powerful cathartic medicines. This condition of the bowels is found to prevail more generally in the severer and more concentrated forms of the disease. In all the epidemics by which our city has been visited, this tendency to constipation has been noted, and is dwelt upon by all our medical writers as an almost constant and obstinate attendant.¹ Nor is it here alone that the symptom has been observed; it has ever formed a characteristic of the fever in other parts of this country, from Boston to Texas.² It has not been found less so in Italy and Spain, including Gibraltar,³ as well as throughout tropical regions.⁴

Costiveness, though universal in some epidemics, and presenting itself very generally in all, is, like every other characteristic symptom, liable to exceptions. It prevails, as already stated, more commonly in the severer and concentrated forms of the disease. But, in some cases of these, the bowels are found to be easily moved by medicine, or even to be naturally affected by looseness or slight purging; and, in a larger number of instances of the more tractable or milder grades, the bowels are, from the outset, amenable to the operation of purgatives or injections, or affected with diarrhœa.⁵

The tendency to costiveness varies in different epidemics. In 1805, it was much less noticed than in other seasons, especially in 1803.⁶ More than a century ago, Desportes had noticed at St. Domingo, that in dry weather the disease is more apt to affect the head, and that, when such is the case, the bowels are more obstinately costive than in moist weather (i. 201). When

¹ Rush, iii. 60, 206; Caldwell, p. 81; Currie, p. 21; *Ib.* on Bil. Fev., 219; Cathrall, p. 38; Barnwell, p. 371; Pascalis, *Fever of 1797*, p. 32; S. Jackson, p. 56; Ffirth, p. 28.

² Lining, ii. 415; Shecut, p. 119; Drysdale, i. 134; Gros, p. 9; Girardin, p. 33; Valentin, p. 164; Townsend, p. 146; Merrill, ix. 244, 245; Cartwright, ix. 11; Dickson, iii. 254; Archer, v. 66; A. Smith, xxv. 502; Hill, v. 90; Tully, p. 295; Ticknor, iii. 223; Baxter, xxi. 3; C. Drake, xxi. 134; Revere, iii. 225; N. O. in 1839, p. 332; N. O. in 1819, p. 8; A. Hosack, p. 12; Seaman, p. 9; E. H. Smith, p. 119; Kelly, xiv. 377; Brown, p. 15; Thomas, p. 83; Stone, vi. 563; Barton, p. 11; Randolph, *Med. Repos.*, xxiii. 168; Addoms, p. 9.

³ Jackson, *Fev. of Spain*, pp. 71, 72; Palloni, p. 4; Gillkrest, ii. 271; Fellowes, p. 208; Audouard, pp. 56, 57; Caisergues, p. 166; Velasquez, pp. 10–13; Pariset, pp. 29, 370–3, 391–3; Boyd, in Johnson, p. 299; O'Halloran, p. 79; Pym, pp. 229, 230; Smith, xxv. 42; Rochoux, p. 480; Burnet, p. 314; Jourdan, v. 257–8; Flores, p. 40.

⁴ Bally, p. 219; Maher, p. 848; J. Clark, p. 9; Comrie, xiii. 167–170; Frost, xiii. 30; Dubreuil, viii. 321; Morgan, iv. 4; Dancer, p. 83; Imray, liii. 80; Rufz, p. 13; Levacher, p. 73; Wilson, p. 8; Pym, p. 229; Boyd, p. 203; Doughty, p. 11; Vatable, p. 345; Chisholm, i. 173; Lempriere, ii. 60; Ralph, ii. 66–70; Blane, p. 440; McArthur, pp. 346, 347; Gillespie, p. 38; Jackson, *Tr.*, p. 259; *Ib.*, *Sketch*, i. 67, 69, 87, 105, 109, 141; Pugnet, pp. 335, 336; Barrington, xii. 311, 312; Peixotto, i. 413; Rochoux, p. 283; Caillot, p. 18; Dickinson, p. 126; Evans, p. 256; *Dict. des Sci. Méd.*, p. 335; Dariste, p. 152; Anderson, p. 5; Finlay, p. 14; Jolivet, p. 8; *Fev. of Cayenne*, in 1850, pp. 167–173; Copland, iii. 139.

⁵ Boyd, p. 203; Evans, p. 256; Moultrie, p. 6; Fellowes, p. 53; Gillespie, p. 38; Dariste, p. 162; R. Jackson, *Sketch*, i. 105, 141; Townsend, p. 186; Pariset, pp. 373–391; Tully, p. 295; Bally, p. 219; Valentin, p. 165; Caillot, p. 18; Revere, iii. 225; Stone, vi. 563; Pariset, *Obs.*, p. 29; Rush, iii. 60; Currie, p. 21; Seaman, p. 9; S. Jackson, p. 60; E. H. Smith, p. 119; Rochoux, pp. 283, 479; Rufz, p. 14.

⁶ Caldwell, p. 81.

in the early period or stage of the disease stools are obtained, either naturally or through the agency of art, they are sometimes feculent or natural in colour and consistence.¹ In some instances, indeed, these natural and feculent stools continue throughout the whole course of the disease. But such instances are not always met with; and, during some epidemics, are seldom seen.²

In cases in which the evacuations are free, especially those in which they are frequent, they are rather of a partially dysenteric nature, and not discharges from the whole course of the intestinal tubes. In some instances, they are purely dysenteric, and attended with the usual symptom of that disease.

In some instances—during some epidemics frequently, during others seldom—we find the stools yellowish and coloured with bile, or even purely bilious.³ In many, they are dark-coloured or green,⁴ especially when obtained by medicine. Again, sometimes, though not necessarily, when stools occur spontaneously, they consist in great measure of mucosities, of a light drab, cream-like or purulent-yellow, or whitish-yellow colour, not unlike the evacuations observed in jaundice, or resembling white clay, or dirty gum-water, and indicating a deficiency of biliary secretion.⁵ In other instances, they assume an ash, or grayish colour.⁶ With this, they are often thin and watery⁷—sometimes like the washings of flesh.⁸

At the period of remission, the bowels generally lose the disposition to costiveness—they are more under the control of opening medicines; and, in some cases, especially in those that end favourably, the patient experiences a slight purging of a bilious character. In the after and last stages of the disease, the bowels are more or less freely opened, and in not a few cases diarrhoea sets in, and is attended with a greater or less degree of pain in the bowels. At this time, and towards the last moments, the stools are sometimes passed involuntarily. The matter voided assumes gradually the character of that ejected from the stomach—becoming brown or black, with coffee-grounds, or in substance resembling ground charcoal, floating in a

¹ A. Smith, xxv. 502; Rush, iii. 61; *Ib.*, iii. 206; Velasquez, p. 10; Townsend, p. 146; Rochoux, pp. 283–480.

² O'Halloran, p. 79.

³ Pugnet, pp. 355–6; Smith, xxv. 502; Bally, p. 219; Pariset, i. 373–422, 3; Drysdale, i. 136; Gros, p. 10; Townsend, p. 146; Currie, p. 22; New Orleans, 1839, p. 333; Blin, p. 6; Gros, p. 9; Berthe, p. 86; Audouard, p. 58; Lefoulon, p. 69.

⁴ Imray, liii. 80; McArthur, p. 347; Barrington, xii. 312; Rush, iii. 61; Baxter, xxi. 4; Lining, ii. 423; Gillkrest, ii. 271; S. Jackson, p. 60; Brown, p. 15; E. H. Smith, p. 121; Audouard, p. 58; Campbell, *Fev. of Charleston* (in Watts), p. 250; *Dict des Sci. Méd.*, p. 335; Shecut, p. 120; Blin, p. 6; Stone, v. 563; Louis, p. 232.

⁵ A. Smith, p. 502; Chisholm, i. 174; Savarésy, p. 273; Bally, p. 219; Gros, p. 9; Dariste, p. 163; Rush, iii. 60; *Ib.*, ii. 10, 11; Currie, pp. 21–219; Drysdale, i. 136; Baxter, xxi. 4; Velasquez, p. 10; Valentin, p. 171; R. Jackson, pp. 51–56; Rochoux, pp. 284–480; Rufz, p. 15; Blane, p. 440; Kelly, xiv. 379; Stone, ii. 563.

⁶ Palloni, p. 4.

⁷ R. Jackson, i. 67–69–87–105–9–141; Rush, iii. 207; Currie, p. 21; Ralph, ii. 68; O'Halloran, p. 79; E. H. Smith, pp. 121, 2; Palloni, p. 4; Savarésy, p. 273; Pugnet, pp. 355, 6; Rochoux, pp. 284–480; Stone, v. 563; Deveze, p. 24.

⁸ *Dict. des Sci. Méd.*, p. 337; Bally, p. 243.

serous fluid. In some cases it presents the appearance of molasses, tar, or thin pitch, in smoothness, tenacity, colour, or consistence; of a mixture of starch, and of an ichorous thin fluid like soot, and in many cases contains streaks, or a large admixture of blood, or consists at times of grumous or even of unchanged blood.¹

The black fluid above mentioned has been found to differ in nothing from black vomit. It is often of an intensely acid and acrimonious nature. In one case, mentioned by Dr. Finley, it was so much so as to corrode the delft-pan of a close stool, which had never been used before. The black marks which were literally branded in it could not be got out, though washed frequently in scalding water (p. 16). Under these circumstances, they often excoriate and inflame the anus.

Dark, watery, coffee-ground stools, are most frequently noticed before black vomit sets in, and continue sometimes for two or three days. In other instances, they continue only twelve or fifteen hours. They appear to cease when black vomit has been ejected.² At the same time, and often in the first stage, particularly when obtained by purgatives, the matter discharged has an offensive odour; emitting, indeed, in some cases, an intolerable and dreadful fetor, alike repulsive to the sick and attendants. When, on the contrary, the disease ends favourably, the alvine evacuations, after being of an unhealthy character, become bilious, and gradually assume a natural appearance.³

In some instances, there is a discharge of worms from the bowels; in other instances, from the mouth.⁴

¹ Deveze, p. 24; Moultrie, p. 14; Archer, v. 67; Currie, p. 26; Drysdale, i. 136; Thomas, p. 84; Stone, vi. 563; Kelly, xiv. 378; Dalmas, p. 9; S. Jackson, pp. 60–80; Shecut, p. 120; Gros, p. 11; Girardin, pp. 34–36; A. Hosack, p. 15; Monson, p. 180; E. H. Smith, pp. 121, 2; Bally, p. 243; Caldwell, *Fev. of 1805*, p. 85; Lining, ii. 415; Dickson, iii. 256; Townsend, p. 154; Valentin, p. 171; C. Drake, xxi. 125; Ticknor, iii. 228; Louis, p. 233; Blin, p. 7; Jackson, *Fev. of Spain*, p. 103; Pym, p. 234; Gillkrest, p. 271; A. Smith, xxv. 502; Fellowes, pp. 54, 55; Audouard, p. 61; Caisergues, p. 168; Berthe, p. 86; Boyd (in Johnson), p. 300; Velasquez, p. 11; Pariset, *Obs.*, pp. 30–37; Pariset, pp. 422, 3; New Orleans, 1839, p. 333; R. Jackson, pp. 56–92; Chisholm, i. 166–173, 4; Lempriere, ii. 65–85; McArthur, p. 347; Osgood, p. 12; Imray, liii. 81; Bancroft, p. 32; Hillary, pp. 151, 2; Madrid, p. 25; Gillespie, p. 42; R. Jackson, *Tr.* p. 262; Pugnet, p. 356; Desportes, i. 199; Vatable, p. 346; Blane, p. 411; Belcher, pp. 249–251; Levacher, pp. 73, 4; Bally, pp. 230–245; Caillot, pp. 18, 19; Dariste, pp. 163–5; J. Clark, p. 14; Rochoux, pp. 284–480; Copland, iii. 139; Mabit, p. 10; Anderson, p. 8; Dickinson, pp. 128–131; Arnold, p. 10; Evans, p. 257; *Dict. des Sci. Méd.*, xv. 337.

² Hayne, Charleston J., vii. 11.

³ Rush, iii. 62; *Ib.*, iv. 10; Deveze, p. 26; S. Jackson, p. 60; Bally, p. 243; Kelly, xiv. 378; Lining, ii. 415; Belcher, p. 251; Levacher, p. 73; Imray, liii. 80; Palloni, p. 4; Chisholm, i. 173–4; Bancroft, pp. 33, 4; Madrid, p. 25; Pugnet, p. 356; Gillespie, p. 42; R. Jackson, i. 112; Drysdale, i. 136; Gros, p. 11; Girardin, p. 36; Valentin, p. 171; C. Drake, pp. 135, 6; Caisergues, p. 169; Berthe, p. 86; Velasquez, p. 10; Pariset, p. 30; Arejula, *Ed. Journ.*, i. 448; Baxter, xxi. 4; Townsend, p. 154; Rochoux, pp. 284–480; Dickinson, p. 131; Stone, vi. 563; A. Hosack, p. 15; E. H. Smith, p. 122; *Dict. des Sci. Méd.*, pp. 336, 7; Shecut, p. 120; Waring, p. 47; R. Jackson, *Fev. of Spain*, p. 105.

⁴ Rush, iv. 11; Labat, i. 73; Nassy, 24.

CHAPTER XII.

DIGESTIVE ORGANS CONTINUED—BLACK VOMIT.

WE have seen that, in a large number of cases, to a period of arterial excitement, of longer or shorter duration—usually from 48 to 72 hours—and constituting, as it were, a single paroxysm, there succeeds one of repose or remission, during which the patient, and not unfrequently his medical attendant, flatter themselves with the idea that the danger is over and a complete recovery at hand. But, although in the milder forms of the disease, these expectations are often realized and convalescence soon sets in, experience teaches that, in very many instances, this period of repose, however well marked it may be, proves treacherous, and is followed, not by a recurrence of febrile excitement, as occurs in other forms of autumnal fevers, but by a state of collapse, attended with a series of symptoms of a more or less formidable character, which leads usually to a fatal termination. Among these symptoms, the seriousness of which will not be contested, the ejection from the stomach of a peculiar matter, which, from its ordinary colour and appearance, has been long designated by the name of the black or coffee-ground vomit, stands pre-eminent.

By not a few medical writers, in this and other countries, it has been regarded as among the most distinctive features of the yellow fever. Indeed, there are not wanting those who view it in the light of the pathognomonic phenomenon *par excellence*—that which enables us to distinguish this from every other form of febrile diseases—and who, though not going so far as to assert that none but cases attended with black vomit are to be regarded as entitled to the name of yellow fever, or uniting in sentiment with the advocates of the theory which teaches that, so long as this matter is not ejected, it is impossible to diagnose that fever, think that the occurrence of the symptom is of itself sufficient to remove all difficulty as to the nature of the disease, while, on the contrary, its absence is equally sufficient to produce great doubts in reference to that question. Sir William Pym, in a letter to Mr. Vance, speaking of this black matter, calls it the most fatal “as well as the most distinctly marked symptom of the disease,” and one which, when united with a speedy termination, leaves no doubt as to the real nature of the case.¹ More recently, in a letter to Mr. Greville, of the Admiralty, on the subject of the fever which spread so fatally on board of the *Æclair*, Sir William speaks of the symptom in question as peculiar to the disease, and not encountered in kindred affections;² and in another letter, addressed to the Lords of the Council, on the fever of Boa Vista, he says: “This disease is unknown in the

¹ On Bulam Fever, p. 60; *Ib.* 2d ed., p. 37.

² See the 2d ed., p. 189.

East Indies, in Egypt, or in Turkey, and is a native of, and peculiar to, the west coast of Africa; it is attended with the peculiar and fatal symptom of black vomit, a symptom which rarely, if ever, appears in the marsh or remittent fever."¹ It would be easy to show that other writers, of more or less note, have entertained opinions similar to those of Sir William Pym, relative to the importance of the black vomit in a diagnostic point of view,² and to its being exclusively an attendant on the yellow fever; and it is scarcely necessary to remark that the name of *vomito prieto*, given to the disease by the Spaniards, sufficiently indicates that, according to them, the symptom belongs to no other form of fevers, and serves to impart to the one in which it shows itself a peculiar and specific character—to stamp it as it were with individuality.

But whatever may be the opinion of Sir William Pym and others on the subject, experience has shown the impropriety of carrying it to the extent they have done. When considered by itself, without reference to other phenomena by which it is preceded and accompanied, and especially when noticed only in a single or a few sporadic instances, the black vomit is not sufficient to stamp the disease in which it occurs as being the true yellow fever; while, on the other hand, its occurrence in this disease is not sufficiently constant and necessary to justify us in refusing to recognize as such, cases which present its other symptoms, merely on the ground that black matter has not been ejected from the stomach.

In support of the first of these propositions, attention may be called to the fact that the dark coffee-ground substance in question is not unfrequently the effect of other causes than the peculiar poison giving rise to the yellow fever, and that it occurs at times not only in diseases more or less remotely connected with the latter, but occasionally in cases in no way allied to it. In the description of the Black Disease, contained in a treatise ascribed to Hippocrates, the matter thrown up is spoken of as being black bile, or as resembling lees of wine; at other times, as a matter resembling blood. "Sometimes the matter vomited resembles the second wine; sometimes it is like the ink of the cuttle-fish; sometimes it is acid, like vinegar; sometimes it consists of saliva and thin phlegm; sometimes of greenish bile. When the black blood-like matter is vomited, it smells like putrid or sanious blood. The fauces and mouth are scalded by the acrimony of the vomit; it sets the teeth on edge, and effervesces with the earth on which it falls; when the vomiting is over, temporary relief ensues." We shall see, as we proceed, that much of what is here said by the father of medicine will apply very accurately to the true black vomit of yellow fever.

In common melæna, the general symptoms of which do not correspond to those of yellow fever, blood in various gradations of change, from a red fluid to a dark matter resembling the grounds of coffee, has been sometimes ejected from the stomach.³ In the description of the variety of the disease which

¹ Parliamentary Doc., p. 5.

² Shecut, p. 120.

³ Bancroft, p. 31; Portal, Observations sur la Nature et le Traitement du Melæna; Mém. sur Plusieurs Maladies, ii. 129; and Mém. de la Soc. Méd. d'Emulation, ii. 107.

was denominated melanosis by the older writers, we are told that "the patient is suddenly seized with vomiting of dark-coloured blood, together with a discharge, by stool, of blood of the same appearance, or more frequently of a very dark and often extremely fetid semifluid mass, of the consistence and colour of tar. Occasionally, the black matter discharged is mixed with blood of more unequivocal appearance."¹

Black vomit has not unfrequently resulted from the action of various poisons. That such is the case in regard to arsenic, we have the testimony of Sauvages,² of Dr. Edward Miller,³ Dr. Waring,⁴ and Dr. Shecut.⁵ Poisoning by corrosive sublimate and verdigris has been known to result in the ejection of a similar matter from the stomach.⁶ Dr. Monges mentions an interesting case in which similar effects followed the ingestion of a large dose of carbonate of potash.⁷ I have known a very analogous effect produced by a quantity of borate of soda, swallowed by mistake, the patient becoming jaundiced and throwing up more than a pint of black matter, bearing a close resemblance to that ejected in the last stage of yellow fever, to say nothing of pain, fever, præcordial distress, &c. Vegetable poisons of various kinds are reported by good authorities to have occasioned like effects.⁸ We know, also, from olden times, that animal poisons, those of the viper, scorpion, &c., produce occasionally effects of the kind noted above.⁹ It was long ago found, and has recently been insisted upon by Dr. Mitchell, of this city, that fungi of various kinds possess the power of producing phenomena somewhat akin to those characteristic of malarial fevers generally, but more particularly to those of the yellow fever; and among them figures the black vomit.¹⁰ The same symptom has been known to arise from mechanical injuries to the stomach. Dr. Monges mentions a case in which it was produced by the kick of a horse.¹¹

The ejection of a similar fluid from the stomach or bowels is often found to follow the introduction of putrid substances into the circulation. Levacher states, as the result of his experiments on dogs, in which, by the way, the act of vomiting is not easily excited, that the discharge of bilious and porraceous matters from the stomach was soon succeeded by that of blood, and of

¹ Carswell, *Cyclop. of Pract. Med.*, iii. p. 80, &c. Portal, *Mémoires sur Plusieurs Maladies*, ii. 129; *Trans. of Irish College of Phys.*, i. 124; *Dublin Hosp. Rep.*, i. 259.

² Sauvages, *Nosologie Méthodique*, iii. 112.

³ Works, pp. 152, 153; *New York Med. Repos.*, ii. 412.

⁴ Report on Yellow Fever of Savannah in 1820, pp. 37, 38.

⁵ Essays, p. 113.

⁶ Miller, *op. cit.*, pp. 51, 57; Chaussier, *Consult de Med. Leg.*, p. 40; Caillot, *Fièvre Jaune*, p. 84; Percival's Essays, ii. 122.

⁷ *North American Med. and Surg. Journ.*, ii. 60.

⁸ Hunter, *Dis. of Jam.*, p. 156; Wepper, *Hist. Cicutæ*; Miller, *op. cit.*, p. 153; Dewitt on Stramonium, *Med. Repos.*, ii. 30; Sauvages's *Nosol.*, iii. 444; Rochoux, pp. 79, 80; Salva, p. 172, quoted by Rochoux.

⁹ Sauvages, iii. 112, 115, 444; Ferguson, *Recol.*, pp. 204, 205; Caillot, *op. cit.*, p. 296; Bally, *Typhus d'Amérique*, p. 552; Levacher, *Guide des Européens aux Antilles*, p. 78; Hunter, p. 156.

¹⁰ Sauvages, iii. 115; Mitchell on the Cryptogamic Origin of Fevers, p. 73.

¹¹ *Op. cit.*, p. 60.

a fluid which he likens to that thrown up in the yellow fever. Then follow dysenteric and black stools.¹ It should be borne in mind that Levacher, who cites these facts as illustrative of the close analogy existing between the yellow fever and diseases produced by putrid and poisonous substances, was perfectly familiar with that fever, and as able as any physician to decide the question of similarity between the fluids vomited in both cases.

Hear what Magendie says on the subject: "No sooner have a few particles of putrid matter passed into the circulation, than the animal is attacked with formidable symptoms. He throws up by vomiting a blackish and grumous (*poisseux*) liquid. This," Magendie adds, "is nothing more than blood exhaled from the inner surface of the stomach, the mucous membrane of which, throughout its whole extent, is puffed up by deposits of blood effused beneath it and in the cellular tissue."²

The second and third volumes of Magendie's *Journal of Physiology* contain accounts of experiments by M. Gaspard, confirming the foregoing statements. The facts resulting from these experiments evidently point, as is remarked by Magendie, to the cause of the black vomit, which takes place in yellow fever, certain typhus cases, &c. A much higher authority than the French physiologist, so far as respects the black vomit of the former of these diseases—the late Dr. Harrison, of New Orleans—remarks, in relation to the result of the aforesaid experiments: "No one, I think, can fail to be struck with the extraordinary resemblance of these symptoms and *post-mortem* lesions to those of yellow fever. The characteristics of the disease, its rapid course, its hemorrhagic tendency, its peculiar lesions, are all to be met with in these experiments. We have *black vomit*, bloody alvine discharges, &c., all so characteristic of yellow fever."³

Dr. Evans relates the case of a woman who died of metro-peritonitis, occurring in the puerperal state, and arising, apparently, from the putrefaction of the retained placenta. The abdomen was *ballonné*, and exceedingly painful to the touch. There were fetid discharges from the vagina, with great heat and pain. The pulse was scarcely perceptible, and the extremities were cold. "But the most remarkable symptom was the almost incessant vomiting, without effort, of immense quantities of a dark, chocolate-looking matter, filling several chamber-pots." On dissection, the stomach was found to contain "a quantity of black matter, similar to that vomited, probably the greater part of a pint." "In this case," says Dr. Evans, "was observed that symptom which, according to many, constitutes one of the diagnostic peculiarities of yellow fever." And as that physician was perfectly familiar with the characteristic appearances of the black vomit, as observed in the latter disease, we may presume there can be no doubt as to the nature of the fluid ejected in the case above mentioned. Dr. Evans again adverts to this case while treating of black vomit, in a subsequent part of his work.⁴ M. Levacher refers to instances of the same kind, arising from analogous causes (p. 80).

¹ Op. cit., p. 80.

² *Phénomènes Physiques de la Vie*, iii. 295.

³ On the Causes of Yellow Fever, N. O. Journ., iii. 570.

⁴ A Clinical Treatise on the Endemic Fevers of the West Indies, pp. 247, 248.

The discharge of a similar fluid from the stomach or bowels, or its discovery in these organs on dissection, is not unfrequently noted in fatal cases of some forms of puerperal fever. It is often mentioned by the older writers on the disease, and alluded to more or less explicitly in works of modern date. That much of what is therein said of the ejection of black matter may not apply to a substance in every way similar to the coffee-ground vomit of yellow fever, I shall not attempt to deny; for the characteristic features of the fluid observed are not always described with sufficient minuteness and precision to enable us to form an accurate idea of its true nature. In addition to this, it may be remarked, that the matter thrown up in puerperal fever is usually stated to have consisted of altered bile; and though we know, from what has occurred in relation to the yellow fever, that little reliance can be placed on assertions of the kind, the fact that bilious discharges may and do sometimes take place in puerperal fever, must induce us to refrain from adducing the statements of several writers who mention the symptom in proof of the occasional discharge of true black vomit in this disease.¹ Dr. Gordon is more explicit on the subject: "A vomiting of bile of a green colour was a symptom which frequently occurred, especially when the patient was costive; and when there were symptoms of mortification, what the patient vomited was black, and had a strong resemblance to the grounds of coffee."² "Frequent vomiting of a coffee-coloured fluid" is mentioned by Dr. Armstrong, who justly considers it as a highly dangerous symptom.³ Black vomit in puerperal fever is further mentioned by the late Dr. Monges, of this city,⁴ Dr. Dickson, of Charleston,⁵ and Dr. Dickinson.⁶ Dr. Dewees says: "We have seen more than one instance of puerperal fever terminating in black vomiting, similar to that observed in yellow fever."⁷ "I have invariably remarked," another writer states, "that whenever the pain was in the epigastric region, vomiting was more severe and constant. At first, the matter vomited has a yellow or greenish appearance, or consists only of the liquids taken into the stomach; but in the last stage of the disease, the vomiting is incessant, and consists of a dark-coloured matter, like coffee-grounds, resembling in every appearance the black vomiting so common in yellow fever; and in one dissection of mine there was an immense quantity of this fluid, the stomach and all the small intestines being filled with it. Dr. Kellie had a case in which, on dissection, about a gallon of the same dark-coloured fluid was found."⁸

The following extract from a letter addressed to me by Dr. C. D. Meigs, of this city, whose extensive experience in this disease is well known, deserves

¹ Hulme, *Essays on Puerperal Fever*, published by the Sydenham Society, pp. 66, 97; Leake, pp. 184, 189, 194, 195; Clarke, p. 424. See also Robert Lee, *Cyclop. of Pract. Med.*, ii. 252.

² *Essay on the Puerperal Fever*, p. 10; *Essays of Sydenham Society*, p. 450.

³ *Observations on Puerperal Fever*, p. 131.

⁴ *Op. cit.*, p. 60.

⁵ *Essays*, i. 355.

⁶ *Observations on the Inflammatory Endem.*, p. 51.

⁷ *Diseases of Females*, p. 378, note.

⁸ John Mackintosh, *A Treatise on the Disease termed Puerperal Fever*, pp. 42, 43.

attention: "In compliance with your request, I have to state that, during my very long and continued practice as accoucheur in Philadelphia, I have had many occasions to observe the changes that precede dissolution in childbed fever. I think that fatal cases of that disease will rarely fail to be attended with black vomit, beginning in the last stages. In the course of my practice, I have also seen fatal yellow fever attended with black vomit. I have also observed that persons perishing with acute peritonitis, often, in approaching the final stage of existence, are attacked with black vomit. I am fully convinced that, in the black vomit of childbed fever, of yellow fever, and of acute peritonitis, in males or females, no difference whatever can be detected, and that the ejection of that fluid is not, therefore, peculiar to yellow fever." The same highly distinguished physician reverts to the subject in several parts of his late work on puerperal fever, and especially in a graphic and splendid description of a case which came under his observation.¹

In a case of peritonitis, from perforation of the intestine, consequent on typhoid fever, which occurred during the last winter, the patient threw up, in my presence, a large amount of black vomit. He had thrown up some before my visit, and ejected more after I left. He died an hour or two after.

Dr. Dewees states that he has seen the same appearance after rupture of the uterus.² And in another work he remarks: "She (the patient) is sick at stomach, and most frequently vomits. The matter discharged is sometimes the common contents of the stomach, at other times it consists of a very dark, even black-coloured substance, resembling coffee-grounds."³ Another writer, already quoted—Dr. Dickson, of Charleston—is of opinion that black vomit always follows the occurrence just mentioned.⁴ The same gentleman adds: "Dr. P. G. Prioleau, whose professional experience has seldom been equalled, and whose authority upon any point of fact is indisputable, assured me he had repeatedly known it (black vomit) to take place among the early vomiting of pregnant women, without unpleasant results."⁵ In another publication, Dr. D. relates the following case: "On the 17th of November, 1825, I was desired to visit, in haste, Mrs. J. F., a lady in the eighth month of pregnancy. I found her sitting supported in bed, throwing up the black vomit. There were violent headache, severe pain in the eyes, which were intolerant of light; no pain at the epigastrium. A slight purgative was ordered, and she was next day as well as ever."⁶ Dr. Nott, of Mobile, recalls the fact that, when fever occurs to women in the state of pregnancy, and there is much and protracted vomiting, small specks or streaks of black matter, like broken-up butterfly wings, are thrown up, very similar to those which form the usual precursors of black vomit in yellow fever. Dr. Crawford related to him the following case: "The patient, during the month of December last (1844), ill of protracted fever, possessing no other symptom of yellow fever, and at a time and

¹ On the Nature, Signs, and Treatment of Child-bed Fevers, pp. 158, 215.

² Diseases of Females, p. 378.

³ System of Midwifery, p. 464.

⁴ Essays, p. 355; Phila. Med. and Phys. Journ., xiv. 208.

⁵ Essays, i. 355.

⁶ Med. and Phys. Journ., xiv. 207.

place where this disease was not prevailing, threw up black matter profusely, which could not be distinguished from the black vomit of yellow fever."¹ In some cases of fever which occurred in New York during the summer of 1843, and were regarded as bilious remittents, the patients vomited a blackish fluid, with coffee-grounds, designated by some *black vomit*. In a case which was admitted into the hospital, the stomach, on dissection, was found to contain "about two ounces of a brownish-black matter, consisting of small distinct floeculi, mixed with the thick ropy mucus which covered the lining membrane."² In a case occurring at Yonkers, in 1843, the patient had a sense of weight and burning at the stomach, and "threw up, at different times, a pint of dark coffee-ground matter." It was "of a dark-brownish colour, in small granules or floeculi, mixed with, but not dissolved in, mucus, or what had been taken into the stomach, and it had little or no odour."³

Dr. Dickson also has met with the black vomit in several cases of bilious remittent fever. In 1825, he saw two patients die of that disease upon the Charles-ton Neck, who ejected that fluid by the stomach and bowels. In each, the symptoms appeared on the fourth day. They lived at some distance from the city, on low, wet ground, and were particularly exposed to the ordinary causes of autumnal remittents and intermittents. Dr. D. saw cases of the kind in 1827.⁴ The black vomit has been noticed in malignant cases of our lake fevers.⁵ Dr. Hildreth, in his account of the fever which prevailed at Marietta, Ohio, in 1823, enumerates the black vomit among the symptoms of the most severe cases.⁶ The same symptom has been observed in the remittent fever of other parts of this country.⁷ Dr. Fenner, of New Orleans, mentions two cases of the sort as occurring in 1850. The first was that of a boy, eight years old, who arrived there in 1847, and suffered from the epidemic of that year. He afterwards enjoyed good health until about the 15th of July, 1850, when he was severely attacked with dysentery. He was never suspected of having the yellow fever; but, on the night of the 19th of July, he threw up black vomit freely and died. There was no yellowness of the surface nor of the eyes. The other case was a dissipated Irishman, who had been several years in New Orleans. He was admitted into the Charity Hospital on the 11th of September, labouring under the prevailing remittent bilious fever, with great irritability of stomach and copious bilious vomiting. On the day following, the ejecta became darker, with floeculi resembling the beginning of black vomit. There was also hemorrhage of the gums. None of the phenomena observed during life, nor the appearances discovered after death,

¹ Amer. Journ. Med. Sci., N. S., ix. 283.

² Forry, *Rondout Fever*, N. Y. Journ. of Med., i. 340-345.

³ Ibid., p. 338.

⁴ Essays, i. 355; Phila. Med. and Surg. Journ., xiv. 208; American Journ., ii. 73.

⁵ Coventry, *Trans. of Med. Soc. of State of New York for 1825*, p. 46. See also *Second Report of Quarantine*, London, 1852, p. 377; Willoughby, N. Y. Med. and Phys. Journ., iv. 209.

⁶ Phila. Med. and Phys. Journ., iii. 110.

⁷ Cooke, *Med. Recorder*, vii. 507.

were calculated to lead to the suspicion that the patient was affected with the yellow fever.¹

Cleghorn saw the black vomit in the tertian fever of Minorca, the symptoms of which, even in its worst form, bear but a remote analogy to those of the yellow fever. In that disease, he found that "the utmost danger is to be apprehended if black matter, like the grounds of coffee, is discharged upwards and downwards."² In several cases of Mediterranean fever, detailed by Sir William Burnett, and some reported to him by Dr. Ross, the black vomiting took place.³ Though not seen by Dr. McWilliams,⁴ nor by Dr. Pritchett,⁵ in the fever they observed during the Niger expedition, there are not wanting facts to show that it is not always absent in the closing scene of the African remittent. Sure it is that black vomit occurs occasionally in certain malignant or pernicious forms of the fever from which the French troops have so severely suffered in Algeria.⁶ Pugnet mentions vomiting of black matter as a fatal symptom in the Demel-Mouia, a form of pernicious fever observed in Egypt.⁷ The Batavian fever, even that of the island of Edam, for a graphic description of which we are indebted to Dr. J. Johnson, differs in too many respects from the true yellow fever to be confounded with it. It partakes much more of the character of the malignant bilious remittents of many tropical regions. Some of the patients on shore were carried off in eighteen, twenty-four, thirty, or forty hours, but many not till as many days after the attack, especially when removed on board from the more noxious air of the island. "The fatal terminations generally happened on the third, fifth, seventh, ninth, and not unfrequently the eleventh and thirteenth days." If they passed this period, they usually lingered out twenty or thirty days. This prolongation of the disease is sufficient—apart from other phenomena noticed in its course—to establish a line of demarcation between it and typhus icterodes; and yet in a few—not a majority, as in the latter fever—of the cases that ended fatally, the patients threw up a fluid resembling the black vomit. Dr. Johnson, it is true, denominates the fluid "black bilious stuff;" but as he adds that it resembled "the grounds of coffee," and it is doubtful whether bile ever assumes that character, and as, besides, we cannot find that Dr. J. regarded the discharge which takes place in true yellow fever as being other than of a bilious nature, we may justly view the fluid above mentioned as similar to that thrown up in the last-mentioned complaint.⁸ The black vomit is also noted as of occasional occurrence in the Bengal remittent.⁹ Dr. Cameron, in his *Remarks on the Endemic Fevers of Ceylon*, does not mention vomiting of this matter, but says: "The intestines generally present a dusky leaden hue. The stomach

¹ Southern Med. Reports, ii. 89, 90.

² Dis. of Minorca, p. 175.

³ Account of the Mediterranean Fever, pp. 50, 157, 421.

⁴ Med. Hist. of the Expedition to the Niger, p. 138.

⁵ Some Account of the African Remittent Fever, p. 162.

⁶ Haspel, *Maladies de l'Algérie*, ii. 167, 168; Boudin, *Fièvres Intermittentes*, p. 155.

⁷ *Fièvres Pestilentiellles*, p. 234.

⁸ *The Influence of Tropical Climates, &c.*, pp. 130-132.

⁹ Johnson, p. 47.

often contains a dark grumous fluid, the black vomit of the West."¹ Trousseau mentions somewhere a case of pernicious fever in which the black vomit was "perfectly identical" with that which is thrown up by patients labouring under yellow fever. Lancisi records the appearance of brown and black vomit and stools in an epidemic at Pesaro, in 1708. Garnier saw it in the fever hospitals at Rome, and even at Versailles.² In the West Indies, it may, as Dr. Imray informs us, be of rare occurrence in the bilious remittent and seasoning fevers; but that it occasionally occurs, we may infer from the expression he himself uses, and the more positive asseveration of other writers.³ In enumerating the symptoms of the typhus fever he noticed in Dublin, Stoker mentions *black vomiting*, melænous feces, and dusky yellow skin; and adds: "The connection which generally subsists between all those symptoms is evinced by their frequent alternation. Thus, the green and black vomiting often alternate with melænous and alvine discharges; and both of these, in other cases, with petechiæ and dusky yellow colour of the surface of the body, and also with similarly coloured suffusion of the eyes."⁴ In the relapsing fever of Scotland and Ireland, it was occasionally, and during the epidemic of Dundee quite frequently, observed.⁵

The following remarks, in reference to this singular fever, would almost seem to have been intended for yellow fever: "Associated with the yellowness, there are generally depression, less or more delirium, dusky and often porter-coloured urine, black melæna-like stools, and hemorrhages from some of the mucous membranes. In the worst of the cases, black coffee-ground-like matter is ejected from the stomach, and passed per anum. In some cases, the black vomit occurs without the yellowness; and, on the other hand, at the autopsy of yellow patients who have had no black vomit, this matter has been found in the stomach and other parts of the alimentary canal."⁶ And yet, as Dr. Craigie very justly observes (*loc. cit.*), "notwithstanding black vomit and jaundice, it is scarcely possible, with any consistency in nosology and common observation, to admit even the resemblance between this fever and yellow fever."

In the summer of 1822, after a long continuance of unusually hot weather, there occurred in the Hôtel Dieu, of Paris, several cases of fever accompanied with jaundice and black vomit. Two patients were, at the same time, similarly affected at La Charité, in the wards of M. Lermnier, and several instances of what was denominated sporadic yellow fever were seen in Paris. In reference to the patients admitted in that hospital, Andral informs

¹ Edinb. Med. and Surg. Journ., lxxi. 74.

² See Second Report on Quarantine, London, 1852, p. 277; Bulletin of Ac. of Med., xv. 1065.

³ Copland, iii. 144, 1144; Dickinson, p. 133; Second Report on Quarantine, p. 323.

⁴ Pathological Observations, pt. ii. p. 103.

⁵ Craigie, Edinb. Journ., lx. 416; Smith, Edinb. Journ., lxii. 67; Russell, Dublin Journ., viii. 70.

⁶ Cormack, Natural History, Pathology, and Treatment of the Epidemic Fever at present prevailing in Edinburgh, p. 23, 1843.

us that they had delirium, a black tongue, tympanitic bowels, a jaundiced discoloration of the surface, and exhibited evident signs of an ataxo-adynamic state of the system. They both threw up a quantity of a substance bearing a strong resemblance to soot—an appearance which, as we shall see, is often assumed by black vomit. It may be doubted, however, that those cases were anything more than typhoid fever modified by the extreme heat of the season. One of the patients recovered under the use of the most powerful stimuli. The other died, and the autopsy revealed a gastro-enteritis, with red softening of the mucous membrane, and ulceration of the intestines, an anatomical character which does not belong to yellow fever.¹ Magendie mentions, that at about the same period, eleven cases of fever occurred in the Hospitals of Paris, attended with brown yellow colour of the skin, petechiæ, and black vomit.² Long before this, Portal, in his *Essay on Melæna*, already cited, mentioned a case which occurred at Paris, in 1775, and in which the patient threw up a large quantity of black matter, the description of which corresponds with that of the true vomit of yellow fever (p. 174).

The fever which occurred in Copenhagen, in 1788 and 1789, and caused considerable mortality, was characterized by black vomiting. For this reason, and owing to its being attended with yellowness of the eyes and skin, the disease was, and continues by some to be, regarded as the true yellow fever. But, considering that it is stated to have raged for some years before among the sailors of the Danish fleet, which seldom sails to tropical climates; that it did not assume its malignant character before the end of October, 1788, when the fleet was united to the Russian vessels of war, and during the prevalence of cold weather and easterly winds; that it prevailed in the winter; that three hundred patients who were admitted into the Naval Hospital infected many of the attendants, who again propagated the disease to the rest of the town, but especially to individuals connected with the navy; that, indeed, it was contagious; that during the first three days after the removal of the patients on shore, not fewer than six surgeons and sixteen female nurses were attacked by it; that it presented symptoms very seldom if ever noticed in yellow fever, such as gangrene of the feet, hands, face, throat, ears, back; and that it was most successfully treated by emetics, opium, and wine—remedies of doubtful efficacy in yellow fever—we may reasonably demur to the opinion in question, and suspect that, notwithstanding the occurrence of black vomit and jaundice, the disease must be regarded as a modification of ship or typhus fever.³

Dr. Cormack states that a case of typhus fever came under his observation in one of Dr. Alison's fever wards (Edinburgh) in which the patient, on the seventh day of the fever, after a well-marked remission of the symptoms (which had been severe), was suddenly seized with jaundice and black vomit.

¹ Dict. de Méd., 1st edit., xxi. 17, article Typhus.

² Leçons sur les Phénomènes Phys. de la Vie, i. 117.

³ Otto, Med. Top. of Copenhagen; Trans. of the Provincial Med. and Surg. Association, vii. 192. Lond.

The weather was, during the whole of the case, below the freezing point.¹ Instances of typhus fever also occurred in Dublin, during the epidemic of 1827, as we learn from Drs. Stokes² and Graves,³ which, from the circumstances of their being accompanied with yellowness of skin and occasionally with the ejection of black matter from the stomach, were denominated yellow fever. In the description of the symptoms, as given by Dr. Stokes, we find that most of the patients vomited, and that, in two cases, a matter resembling coffee-grounds was discharged from the stomach and bowels. In one case, Dr. Graves found a "considerable quantity of a dark-red fluid in the stomach, with a good deal of a substance resembling coffee-grounds." After what precedes, and considering, too, that these cases occurred during the cold weather of the winter months, when true yellow fever never shows itself, and that the summers of Dublin—supposing cases like those mentioned to have sometimes occurred in that season—are not sufficiently long and oppressively hot to produce that disease, and that the symptoms observed in Dublin are not analogous, the black vomit and jaundice excepted, to those of the fever of this country or tropical climates, we may admit the identity of the fluid thrown up on those occasions, or discovered on dissection, with the true black vomit, without being obliged to admit also the identity of the two fevers, and to coincide with Dr. Graves when he states that "there is not so much difference between the diseases of Ireland and warmer countries as has been imagined;" and that "they differ, it is true, as to their degrees, but not as to their pathology."

Mr. J. Niell, Surg. H. M. S. *Britannia*, relates⁴ a case of fever which occurred at Portsmouth (Eng.) in July, 1827, during a very hot spell of weather. In this case, which was of a severe character, there was jaundice and other symptoms which gave it somewhat the character of yellow fever. The patient was attacked on the night of the 8th; on the 15th he vomited thrice in the night, the matter being black like the grounds of coffee. The stools now assumed the same appearance. He was salivated, and what not; and, after upwards of twenty days of sickness, recovered.

The black vomit has also been found to occur in the oriental plague.⁵ It was observed as an occasional symptom in the Pali plague of India.⁶ Dr. Parry, of Bath, describes a case of local organic disease of the stomach, attended with vomiting of matter very much like the black vomit of yellow fever.⁷ Dr. Dickinson informs us that he has seen "the discharges from the stomach inky, fuliginous, grumous, like coffee-grounds, or containing dark membranous-like shreds, in gastric affections of a purely topical character."⁸

¹ *Op cit.*, pp. 90, 91.

² *Cyclop. of Pract. Med.*, article Enteritis, ii. 59.

³ *Clinical Med.*, 8vo. 1843, p. 215, Am. edit., pp. 186, 192.

⁴ *Lond. Med. and Phys. J.*, N. S., v. 105. Coll., l.

⁵ Clot Bey, pp. 30, 1, 6, 56; Caldwell, p. 19; Pugnet, p. 158; Lacheze, *Bulletin de l'Acad. de Méd.*, i. 351.

⁶ Forbes, Report on the Diseases at Pali, as observed in January and February, 1838; *Tr. of Med. and Phys. Soc. of Bombay*, ii. 24.

⁷ *Medical Notes*, ii. 327.

⁸ *Op cit.*, p. 51.

Dr. Dickson, of Charleston, has also found it to occur in gastritis and enteritis.¹ He has seen it likewise in a case of catarrhal fever, in a child three and a half years old, as also in one of varioloid occurring in winter. It ceased on the appearance of the eruption, and the patient recovered. The same physician saw it in two cases of dropsy. Dr. Prioleau, already mentioned, detailed to Dr. Dickson a singular instance of the occurrence of this peculiar discharge in a healthy boy, without any previous or evident bad symptom, and occasioned by no evident cause beyond mere *fatigue*, the youth having run about and played much on a summer day. He slept after vomiting, and awoke quite well.²

The same discharge has occasionally presented itself in "apoplexy from a stroke of the sun, in injury of the brain, from external violence, and in inordinate intoxication."³ It occurred in this city, a few months ago, a short time before death in a child nine months old (a milc from the district usually infected), labouring under cerebral irritation consequent on common summer bowel affection. Dr. R. Armstrong saw it in a case of psoas abscess, sent to the Hospital of Jamaica, in the last stage of hectic fever. The patient died on the seventh day after admission, from mere exhaustion, and the stomach was filled with black vomit, although none appeared during life.⁴ The writer of these pages has seen an interesting case of copious ejection of well-marked black vomit occurring in a fatal attack of colic. The disease ended fatally in a few hours, and will be alluded to again more in detail in a subsequent part of this volume. Dr. Kelly saw the same kind of discharge at the moment of death from phthisis pulmonalis;⁵ and we are indebted to Dr. R. Armstrong, already mentioned, for the following account of a case of the same kind: "A marine, invalided at Barbadoes, was sent to Jamaica Hospital to wait for a passage to Europe; he was in the last stage of phthisis, with purulent expectoration, hectic paroxysms in the evening, and diarrhoea. On the fourth day after admission, he complained of general indisposition, and loss of appetite; the tongue was clean, and there were no symptoms indicative of any other disease than the affection of the chest. In the evening, he brought up a small quantity of black vomit, became delirious, and died the following morning. On dissection, the stomach was found to contain about a pint and a half of this fluid."⁶

But these examples must suffice. They establish, beyond a doubt, not perhaps the frequent, but certainly the occasional occurrence of black vomit in diseases differing more or less from the yellow fever. It may be that in some of the instances mentioned, occurring as they did in yellow fever regions, and at a period of the year when that disease usually shows itself, the effect will be attributed by some to the influence of the peculiar poison giving rise to the latter. Yet when we take into consideration, that the fever did not prevail at the time, and that the cases in question presented none of its

¹ Essays, p. 355; Phila. Med. and Phys. Journ., xiv. 207.

² Op. cit.

³ Dickinson, op. cit., p. 51.

⁴ Influence of Climate, p. 169.

⁵ Amer. Journ. Med. Sci., xiv., N. S., p. 383.

⁶ Influence of Climate, p. 165.

characteristic symptoms, we can scarcely be justified in explaining their occurrence in that way; while, in other instances, the discharge of black vomit took place under circumstances which render such an explanation perfectly inadmissible, and compel us to refer it to causes totally independent of those of yellow fever.

These facts show, therefore, that the occurrence of black vomit, as already stated, is not by itself sufficient to characterize the disease. Equally certain is it, that other facts demonstrate the impropriety of assigning a different nosological position to, and of refusing to view in the light of true yellow fever, or referring to causes different from those to which this disease owes its origin, cases presenting its well-known characteristic symptoms, on the score of their not being attended with the discharge of the black matter in question. In illustration of the inadmissibility of such a conclusion, appeal may be made to the well known circumstance that, with few exceptions, none of those who recover from the fever throw up coffee-ground matter, which, when it does present itself, is very generally an attendant on fatal cases. Nay, it is not less true, that even among those who die, and especially when the disease ends suddenly or rapidly, or the brain is early implicated, black vomit often fails to show itself.¹ In this city, in 1820, it was computed to have done so in nearly one-third of the fatal cases.² It was frequently absent during the formidable epidemic of Barcelona, in 1821.³ In Gibraltar, in 1814, black vomit was rarely seen.⁴ In the eighty-fourth regiment (British) at Jamaica, in the epidemic of 1827, the black vomit, according to the surgeon's official report, rarely appeared in fatal cases, although the fever was so severe that one hundred and forty men died in six weeks.⁵ Dr. Bancroft remarks, that in cases in which the brain suffers much, and these are not unfrequent, the black vomit does not commonly occur.⁶ Dr. R. Jackson observes, that if the fever terminates before the third day, black vomiting is scarcely ever seen; so, also, when the disease does not go through the usual processes, which is the case in the concentrated form of the fever in persons of a phlegmatic, and of what he calls the gangrenous temperament (the form which prevailed at Grenada in 1793), for in this form the black vomit occurs "on some occasions near the last hours of life; but not often, and only when the vascular action has been excited to a considerable extent in the preceding course of the disease."⁷ During the epidemic which prevailed on board the French frigate *l'Hermione*, at the Havana, the black vomit was very frequently absent; for in three hundred and twenty-three cases which fell under

¹ Bone on Yellow Fever, p. 4; Lewis, New Orleans Journ., iv. 159.

² Jackson, an Account of the Yellow or Malignant Fever which appeared in the City of Philadelphia in 1820, p. 56.

³ Rochoux, Recherches sur les différentes Maladies qu'on appelle Fièvre Jaune, p. 552.

⁴ Cyc. of Pract. Med., ii. 272-3.

⁵ Ibid., p. 272.

⁶ Essay on the Yellow Fever, p. 13.

⁷ A Sketch of the History and Cure of Febrile Diseases, i. 94.

the observation of Mr. Maher, that symptom was only noticed seven times.¹ In New York, in 1822, the black vomit was also of comparatively rare occurrence. In two hundred and fifty-four cases which ended fatally, this symptom appeared only in eighty or ninety.² In 1819 it was equally rare.³ At Natchez, in 1823, it was only a casual symptom, occurring most commonly in the congestive form of the disease.⁴ In Gibraltar, in 1828, according to Dr. Gillkrest, of one hundred and ninety-six cases, only six vomited it during life. Dr. Davy, in a note to Dr. Blair's excellent *Essay on the Yellow Fever*, alludes to an epidemic at Barbadoes, in 1811, in which the gastric symptoms were very inconsiderable. And the same absence of it, though not always to the same extent, has been noticed in other cities of this country, in the West Indies, and on board of infected vessels.⁵

It is to be remarked, that in many of the fatal cases in which the matter of the black vomit is not ejected from the stomach, it may sometimes be detected in that organ during life by the swelling of the abdomen and the gurgling noise heard on motion.⁶ More frequently it is discovered on dissection.⁷

Nor is it less to be borne in mind that considerable difference is found to exist as regards the frequency of this symptom, in different places, or at different times in the same place. While, as we have seen, it occurred seldom on some occasions in the West Indies; at Dominica, in 1838,⁸ and at Guadeloupe, during several years,⁹ it was found in a large majority of fatal cases. At Galveston, in 1839, it was of frequent occurrence, and indeed constant in bad and fatal cases.¹⁰ During the epidemic of 1839, at New Orleans, the black vomit was less frequently noticed than it had been in former seasons,¹¹ but in 1844, it again showed itself in a large number of cases, and, indeed, was proportionally more common than heretofore.¹² Dr. Blair informs us that at Demerara, in 1837, the mortality amounted to four hundred and four out of two thousand and seventy-one cases. Of those who died, three hundred and sixty-six had the black vomit.¹³ While in New York, as we have seen, the discharge occurred but rarely, even in fatal cases; we learn that,

¹ Annales Maritimes for 1842, pp. 847, 850.

² Townsend, pp. 163-4; Pascalis, J. Méd. de Marseilles, v. 140.

³ C. Drake, Med. Repos., xxii. 136.

⁴ Merrill, Phila. Med. and Phys. Journ., vii. 245.

⁵ Irvine, p. 30; Valentin, p. 27; Archer, Med. Rec., v. 68; Baxter, Med. Repos., xxi. 3; A. Smith, Am. Journ. Med. Sci., xxv. 503; Stone, New Orleans Journ., ii. 553; Dickson, Eclectic Journ., iv. 110; Evans on Yellow Fever, p. 257; Osgood, p. 12; Bally, Typhus d'Am., p. 240; Blair, p. 82; Pym, 2d ed., p. 81; Minutes of the Proceedings relative to the Fever which occurred on board the Macedonia, p. 29.

⁶ Dickson, Essays, p. 350; Wood, i. 301.

⁷ Michel, Charleston Journ., v. 748; Townsend, p. 190; R. Armstrong, p. 169; Louis, p. 218; S. Jackson, p. 56; Dickson, Chapman's Journ., iii. 231; Ticknor, N. A. Journ., iii. 228; A. Smith, Amer. Journ. Med. Sci., xxv. 503.

⁸ Edin. Med. and Surg. Journ., liii. 82, 3.

⁹ Rochoux, op. cit., p. 552.

¹⁰ A. Smith, Amer. Journ. Med. Sci., xxv. 503, and Essay, p. 15.

¹¹ Rept., pp. 160, 325.

¹² New Orleans Journ., vii. 43.

¹³ Pages 49, 82.

in former epidemics, the number of cases in which it was observed amounted to full one-half of those that died.¹ In Charleston, it was comparatively seldom encountered in 1817, 1824, and 1827; while in 1835 it was frequent.²

From all that precedes—from the fact that the ejection of black vomit is observed, occasionally at least, in other and diversified complaints, and, on the other hand, that it fails to present itself in a large number of cases in regard to the true nature of which there can be no difference of sentiment, even in many of those that terminate fatally—we cannot, as already stated, regard it as sufficient by itself to characterize the disease as true yellow fever, or coincide in sentiment with those who draw a line of demarcation between cases otherwise presenting the same or kindred phenomena, founded on the occurrence of the discharge in some and its absence in others. But, while entertaining these views, every one conversant with the disease from personal observation, and who has made himself acquainted with the results obtained in various parts of yellow fever regions, whether in tropical or temperate latitudes, is aware that the black vomit occurs much more frequently in true yellow fever than in any other form of febrile or other complaints. Indeed, though, strictly speaking, rather a peculiar termination than a symptom of the disease, since it does not occur till late in the attack, and even then only in a certain category of cases, yet these, owing to the malignancy and usual fatality of the disease, are so numerous, and the occurrence of the discharge among them so common, that the black vomit may be regarded not strictly as pathognomonic of the disease, but as a highly useful diagnostic sign, so common that the word yellow fever brings up to the mind the idea of black vomit, and *vice versâ*. Indeed, there can be no doubt that when it presents itself in more than a few isolated cases, and particularly when it is associated with other symptoms clearly appertaining to the fever, or when, in the absence of these, it occurs during the existence of an epidemic, it stamps the disease with a character which prevents it from being mistaken for any other, and affords us one of the most useful means for drawing a line of demarcation between cases in which it is observed, and others which prevail at the same time, but run their course to recovery or death without exhibiting it.

Not only is the black vomit a very frequent attendant on yellow fever during life, but it is almost constantly found after death in the cavity of the stomach, of the duodenum, or of some portions of the intestinal tube. We have already seen that it is not unusually found there, especially in the former organ, in cases in which it was not ejected during the course of the disease, and it seldom happens that the same result is not obtained when such ejection has taken place. It is recorded by almost every writer who has devoted attention to the pathological anatomy of the disease; by some of whom it is represented as of very ordinary, by others as of constant occurrence. Dr. Blair remarks that black vomit in the stomach is the rule, its absence the exception.³ Dr. A. Smith, of Texas, met with it in all the autopsies he

¹ Townsend, pp. 164, 194.

² Dickson, Eclectic Journ., iv. 110.

³ Page 102.

made, with, perhaps, a single exception.¹ Dr. Webb says: "I think I was present at more than one hundred *post-mortem* examinations of subjects who died of yellow fever at Barbadoes. The stomach was distended with gas, and contained more or less black vomit. I do not recollect having been present at the opening of any body when black vomit was not found."² And other investigators, whether in this city, during the various fearful epidemics by which it has been visited, from 1793 to the present year, in other parts of the United States, in Europe, in the West Indies, the southern coast of this country or Africa, have made similar statements.³

The black vomit usually makes its appearance at the opening, or about the middle of the second stage of the disease; sometimes at the decline of the first stage; occasionally, but rarely, during the first or febrile paroxysm. From this, it will be easily perceived by those who are aware of the ordinary duration of those stages which, though varying somewhat in different cases and in different seasons, do not do so very materially, that the most usual period of its appearance is about the fourth or fifth day. In some instances it occurs earlier, and sometimes later. It has, in some cases, as Dr. Dickson and others remark, shown itself within twenty-eight or thirty hours from the commencement of the attack,⁴ or even sooner. The second day is not unfrequently mentioned as that of its appearance.⁵ On the other hand, we find it sometimes retarded as late as the seventh, eighth, or ninth day, or even much beyond.⁶ The following table, showing the number of cases out of a

¹ Trans. of N. Y. Acad. of Med., i. 64.

² Second Report on Quarantine, p. 366. Lond., 1852.

³ Physick, Med. Repository, v. 130; *Ib.*, in Rush, ii. 92; Ffirth, pp. 36, 37; Bache, Am. Journ., N. S., pp. 121, 2; Caldwell, Fever of 1805, p. 99; Parrish, Med. Museum, iii. 88; Lowber, *ib.*, v. 19, 20, 21, 22; Lawrence, Phil. Med. and Phys. Journ., x. 4, 7, 9, 10, 11, 258; Kelly, Am. Journ., N. S., xiv. 282; Harrison, N. O. Journ., ii. 139; Waring, p. 49; Nott, Am. Journ., N. S., ix. 280; Valentin, p. 180; Gros, p. 19; Chabert, p. 13; Fever of New Orleans in 1820, p. 10; Ticknor, North Amer. Journ., iii. 229; Heustis, Topog. of Louisiana, p. 22; Dickson, Philad. Med. and Phys. Journ., iii. 256; *Ib.*, xiv. 206; A. Smith, Am. Journ., xxv. 504; Hicks, N. O. Journ., v. 221; Slade, *ib.*, i. 86, 94; Hayne, Charleston Journ., vi. 342, 628, &c.; Townsend, pp. 190, 191; Dalmas, p. 15; S. Jackson, p. 56; Arejula, pp. 418, 426, 427; Louis, pp. 80, 81, &c.; Pariset, p. 418; Palloni, p. 11; Burnett, p. 243; Fellowes, p. 68; Vance (in Pym), p. 6; O'Halloran, pp. 189, 192; Audouard, pp. 151, 152; R. Jackson, Fev. of Spain, p. 88; Imray, Ed. Med. and Surg. Journ., liii. 82, 83, 90; *Ib.*, lxiv. 330; Copland, iii. 161; Blair, p. 92; Arnold, pp. 317, 311; Osgood, p. 12; Vatable, p. 348; Gillespie, p. 70; Pugnet, p. 363; Ralph, Ed. Med.-Chir. Trans., ii. 80; J. Clark, p. 19; Catel, p. 12; McArthur (in Johnson), p. 349; Frost, Med. Repos., xiii. 263; Hunter, p. 161; R. Jackson, Sketch, i. 79, 114; *Ib.*, Treatise, p. 265; Dubreuil, Journ. Universel, viii. 329; Caillot, p. 167; Kéraudren, p. 7; Savarésy, p. 461; Hacket, Med.-Chirurg. Rev., xvi. 290; Evans, pp. 215, 216; Cyclop. of Pract. Med., ii. 275; Rufz, p. 18; Bally, p. 194; R. Armstrong, p. 169; Maher, pp. 845, 884; Grant, p. 47; Bone, pp. 26, 27; Bancroft, p. 24; Belcher, Ed. Med. and Surg. Journ., xxiii. 251; Furlong, Med.-Chirurg. Journ., xxv. 290; Boyle, p. 234; Rochoux, pp. 351, 532; Dict. des Sci. Méd., xv. 341; Levacher, p. 76; Heastie, p. 22.

⁴ Dickson, Am. Journ., ii. 73; Kelly, *ib.*, xiv. 384.

⁵ Imray, Ed. Med. and Surg. Journ., liii. p. 83.

⁶ Bone, p. 3.

total of three hundred and sixty-six, in which the symptom was observed on different days of the disease in Demerara, during the epidemic of 1837,¹ will apply with sufficient exactness to other places and other seasons, to be used in illustration of the subject before us:—

Day of the disease	1	2	3	4	5	6	7	8	9	10	11	12	13	Uncertain.	Black vomit doubtful.
No. of cases . .	2	5	32	76	89	83	26	15	10	7	2	2	1	5	11
Rate per ct. . .	0.09	0.24	1.72	3.67	4.40	4.10	1.25	0.72	0.48	0.31	0.09	0.09	0.04	0.24	0.52

In Charleston, in 1854, black vomit occurred sometimes thirty-six hours after the first invasion of the fever. In other cases, it did not come on until the fifth day.²

The black vomit has, from the earliest period in the history of the yellow fever, been viewed by professional and unprofessional observers, as a highly unfavourable sign. It is more so than the jaundiced condition of the skin, being, in that respect, according to Dr. Blair, who has paid much attention to the subject, in the proportion of 75.68 to 46.23. Indeed, the ejection of black matter from the stomach, or its effusion in that cavity, has usually been found to portend, with almost unerring certainty, the approach of death. Such has been the case in some of the epidemics of our city, as also in those of other parts of this country, Europe, and tropical regions, when the discharge upwards of even a small portion of the fluid has proved with few, if any exceptions, the precursor of a fatal issue.³ In other places and seasons, perhaps also in the hands of other physicians, the black vomit has not been found so universally to indicate an unfavorable issue. At all times, and even during the most fearfully malignant visitations of the disease, a few cases present themselves in which the patients escape after throwing up the justly dreaded matter; while, on some occasions, the number of recoveries, notwithstanding the advent of the discharge, is comparatively large. Vatable says, in reference to the epidemic of Guadaloupe in 1826: "We have seen men leave and rejoin their regiments (corps) who, during several days, had thrown up the matter of black vomit."⁴ Dr. Ralph saw three cases of recovery among the sick of his regiment at Barbadoes in 1817.⁵ Dr. Dickson remarks that the black vomit is not always fatal;⁶ and, in another publication, states that in 1827, at Charleston, the number of recoveries after the

¹ Blair, p. 9.

² Wragg, Charleston Journ., x. 82.

³ Rush, iii. 61; Ib., iv. 34; Currie, p. 39; Caldwell, Fever of 1805, p. 85; Parrish, Med. Mus., iii. 192; S. Jackson, p. 56; Lining, ii. 429; Ashbel Smith, p. 32; Shecut, p. 120; Merrill, N. O. Journ., viii. 6; Ib., Memphis Record, iii. No. 4; Gros, p. 17; Fever on board the Macedonian, p. 29; Hayne, Charleston Journ., vii. p. 10; Rouppe, pp. 408, 411; Imray, Ed. Journ., liii. 82; Ib., lxiv. 329; Desportes, i. 199; Lempriere, ii. 68; Rochoux, p. 280, &c.; Evans, p. 248; Valentin, p. 168; Bally, p. 279; Musgrave, Med.-Chir. Trans., ix. 134; R. Jackson, i. 171; Palloni, p. 8; Pariset, pp. 448, 449; R. Arnold, p. 321; Ferguson, Recol., p. 125; Catel, p. 11; Anderson, p. 7; Audouard, p. 7.

⁴ Ann. Mar., p. 350. 1827.

⁵ Edinb. Med.-Chirurg. Journ., ii. 73.

⁶ Phil. Med. and Phys. Journ., xiv. 206.

discharge was great beyond all former example.¹ Five per cent. of black vomit cases, as we are told by Dr. Lewis, recovered at Mobile in 1843.² In another place, he states that "fourteen patients were saved" that year "after the appearance of this usually fatal symptom."³ In reference to the epidemic of Charleston, in 1852, Dr. Cain writes to me: "There have been quite a number of recoveries after black vomit in this epidemic." Of fifty-nine cases which recovered on that occasion in the city ward of Roper Hospital, under the care of Drs. Huger and Post, two threw up black vomit, and nine got well after passing it copiously by stool.⁴ In 1854, out of 74 cases, in which the fluid was thrown up, 9 recovered.⁵ In his account of the epidemic of 1793, Dr. Rush states that many recovered who had thrown up the black-coloured matter. Of 366 cases of black vomit mentioned by Dr. Blair as occurring at Demerara in 1837, out of 2,071 cases of the disease, only 277 died, giving the centesimal mortality stated above, viz: 75.68.⁶ "Fifty-five soldiers affected with the gastric disease called yellow fever, Dr. Bone says, were received into the Naval Hospital, Barbadoes, in 1821; 17 of these vomited; 33 did not vomit; 11 of them died; and of these, 6 vomited and 5 did not vomit."⁷ Cases of recovery after the discharge of black vomit were not unfrequent in the yellow fever which has lately prevailed epidemically on the coast of Brazil. Three occurred on board of a single ship, the Tweed. Others in another English vessel, the Cormorant.⁸ In the fever of Pernambuco in 1850, Dr. Arbuckle says he saw ten persons recover after black vomit.⁹

Dr. Fenner thinks that when well-marked black vomit occurs, the chance of recovery is not much over one in one hundred. In 1853, he had two recoveries in his private practice and several in his wards at the Charity Hospital. A patient of the late Dr. Hester threw up black vomit copiously during twenty-four hours, and recovered. There were fifteen recoveries from black vomit in the Maison de Santé (where the number of cases was 338), according to the resident surgeon, Dr. Boyer. Professor Jones had two recoveries in private practice, and eight or ten at the Charity Hospital. Drs. McKelvey, Lindsay, M. Dowler, Wedderburn, and Rushton, had some twenty more; and Dr. Chappin, house surgeon of the Charity Hospital, gives it as his opinion that seven per cent. of the cases of black vomit in that institution recovered. Dr. F. states that there were more recoveries from it during that season than was ever known before.¹⁰

"Mr. Amiel, who witnessed three yellow fever epidemics at Gibraltar, states that he met with two instances in children, where recovery took place after the appearance of black vomit. Surgeon Collow, of the 84th regiment,

¹ Am. Journ., ii. 73.

² New Orleans Journ., iv. 159.

³ Ibid., vol. i. 300.

⁴ Simons, Charleston Journ., viii. 368.

⁵ Wragg, ib., x. 82.

⁶ Blair, p. 82.

⁷ Bone, p. 4.

⁸ Dr. W. McKinley, Edin. Monthly Journ., Oct. 1852, pp. 335, 337; Lallemand, op. cit., p. 98.

⁹ Report of New Orleans San. Com., 1853, p. 197.

¹⁰ Fenner, Fever of 1853, pp. 53, 54.

says, in his official report to the Army Medical Board, relative to the yellow fever, as it appeared in his corps at Fort Augusta, Jamaica, in the year 1827, that the black vomit 'is not invariably fatal; examples, more than one, in my regiment, are now living.'" Dr. Bone, deputy-inspector of hospitals, who has had an experience of many years in the West Indies, is very precise upon this point, in an official report relative to an epidemic which prevailed at Barbadoes, in 1821. He there not only refers to some cases in which recovery took place after having vomited black ("China ink-coloured") fluid, as well as flaky brown blood, usually (according to him) the precursors of the real black vomit, but enters into minute details relative to two cases ultimately terminating favourably, when the fluid ejected possessed the most unequivocal characters."¹

In the Havana, as we learn from Mr. Maher, it was found by Dr. Belot, whose experience in the yellow fever, obtained in private and hospital practice, is very ample, that while the brownish matter, containing flocculi, is invariably an indication of mortal issue, the chocolate-coloured matter, which, like the former, passes under the name of black vomit (and which corresponds to the China ink-coloured matter, mentioned by Dr. Bone, and considered by him as the precursor of the real black vomit), is not necessarily so.² Dr. Rufz met with one instance of recovery, at Martinique, in 1838, after the occurrence of real black vomit.³ At Barcelona, some few recovered,⁴ and cases of a similar kind are alluded to by Alexander Hosack,⁵ E. H. Smith,⁶ Desportes,⁷ Harrison,⁸ R. Jackson,⁹ and Catel.¹⁰

It may be remarked that, in reference to the degree of importance to be attached to the black vomit in a prognostical point of view, much depends on the class of individuals affected, the peculiar appearance and physical properties of the fluid, and the quantity in which it is ejected. According to Dr. Fenner, black vomit and other hemorrhages rarely occur amongst full-blooded negroes, though he has heard of a number of cases, and in 1853 had one himself. Mulattoes and quadroons are almost as liable to them in yellow fever as white people, and quite a number have died of hemorrhages that year. Black vomit is quite common amongst white children with yellow fever, but they are far more apt to recover from it than adults.¹¹ We have seen that, according to Dr. Bone, recovery may take place so long as the discharge consists of China ink-coloured fluid, as well as flaky brown blood, and that Dr. Belot entertains hopes so long as the patient throws up only a chocolate-coloured matter. Much to the same effect are the remarks of Dr. Lewis, of Mobile, who found that so long as the black vomit is raised in small quantities, thick and pasty, and is thrown up with a natural mucus,

¹ Cycl. ii. 273, 274.

² Annales Maritimes, p. 843.

³ Report by Dr. Chervin, p. 16.

⁴ Audouard, F. J. de Barcelonne, p. 64; Periodico de la Soc. de Salud. Publica, p. 265.

⁵ Essay on Yellow Fever, p. 15.

⁶ Webster's Collection, p. 120.

⁷ Maladies de St. Domingue, i. 222.

⁸ N. O. Journ., ii. 135.

⁹ Fever of the South Coast of Spain, p. 121.

¹⁰ Annales Maritimes, iv. 1844, pp. 225, 6.

¹¹ Fever of 1853, p. 51.

there is some hope of recovery. When, on the other hand, the fluid is thin and black, and contains the coffee-ground sediment, its appearance should always be viewed as a sign of fearful omen.¹ Dr. Fenner says, after alluding to the disgorgement of vessels by black vomit: "Hence, we may explain a fact that has been observed by many, viz: that recovery is more apt to take place after free discharges of black vomit than after very slight." On this subject, I cannot but believe that Dr. Fenner stands alone.

In cases of recovery, the discharge is sometimes protracted a very long while, and even in some cases continues amid a train of phenomena, with which we could scarcely have anticipated to find it associated. In a case stated by Dr. Logan, and referred to by Professor Dickson, of Charleston, black vomit was thrown up during the convalescence of a juvenile patient, whose recovery did not seem at all retarded by the circumstance or its cause. In one case, under the care of Dr. Prioleau, the patient lived after ejecting the black vomit for fourteen successive days.² I have heard of a case in which the discharge continued, though in very small quantities, for twenty-one days. The patient recovered. These, however, are unusual cases. More commonly, convalescence is soon established, and the discharge ceases. In a few fatal cases, the patient lingers a few days after the appearance of the black vomit. One, mentioned by Dr. Dickson, lived ten days after its occurrence. But, in the majority of instances, life is not long protracted. The patient is sometimes carried off in less than twenty-four, sometimes in two or three hours.

In some fatal instances, the discharge of black vomit comes on unexpectedly, and is almost immediately or soon after followed by symptoms of impending dissolution. During the months of July, August, and September, 1853, this was found to occur in several of the cases which fell under my personal observation. The patients, after appearing to be doing well, during the deceptive period of the metaptosis or remission, or presenting few symptoms portending immediate danger, suddenly threw up more or less of the matter, and were soon attacked with convulsions or other phenomena of like import.

In other instances, the first effect of the ejection seems to be of the most beneficial kind. On its occurrence, the feelings of the patient improve; some of the more unpromising symptoms mend; the restlessness diminishes; the inability to take food, the tension and distress at the epigastrium and abdomen generally subside; the tongue assumes a cleaner and more healthful appearance, and the patient often experiences a singular tranquillity of body and mind.³ Dr. Blair, who has in an especial manner pointed out these circumstances, remarks that when, with sudden improvement of the tongue and other symptoms, there exists dirty or other discoloration of the surface, and at the period of the disease when black vomit might be expected, then succussion will sometimes detect the presence of the effused fluid before vomiting occurs.⁴ But, whether the detection be made or not, effusion, under these

¹ New Orleans Journ., iv. 159.

² Dickson, Am. Journ., ii. 73.

³ Currie, Fever of 1793, p. 26; Lewis, New Orleans Journ., iv. 159; Daniel, Fevers of Savannah, p. 72.

⁴ Op. cit., p. 80.

circumstances, may justly be suspected or feared at the time, or very soon after, unless those changes be accompanied by others of a favourable character; the connection between the effusion and the sudden amendment in question being, as already stated, frequently noticed.

The discharge of black vomit, contrary to what might have been anticipated, is not necessarily attended with debility of mind and body. So far from this, experience in this city and elsewhere teaches that, in not a few cases, the retention of the muscular power and intellectual functions, self-possession, and courage, is remarkably great, at a time, too, when every other symptom portends the approach of death. This occurs often in the last stage of the disease, when the latter has run its usual course, and also in what has been denominated walking cases, in which the inflammatory or pyrexial stage is wanting, and the disease, while presenting the remarkable feature in question, passes at once to the malignant stage. Dr. Harrison remarks that "some will even get out of bed and walk about, declaring they are perfectly well, and wish to dress themselves."¹ The following statements will serve to illustrate this. The first, for which we are indebted to Dr. Dickson, is interesting: "I saw a patient in our Marine Hospital, in 1817, walking from ward to ward with a vessel under his arm, to receive the black vomit which he threw up from time to time. He continued this practice for ten days, until within fifteen minutes of his death, which took place suddenly and altogether unexpectedly to the poor fellow; he had always expressed the most cheerful and confident expectation of recovery, notwithstanding this usually much dreaded symptom."²

A man came into the office of a physician in New Orleans, and said that his friends had persuaded him to consult a physician, although he himself believed there was no occasion for it; whilst conversing, he asked for a basin, and threw up a large quantity of black vomit.³ Dr. Townsend, in his *Essay on Black Vomit*, mentions the case of an individual who walked about two hours before his death, while vomiting up the fluid in question (p. 5). Dr. Dashiell relates a case in which it unexpectedly appeared whilst a man was getting shaved in a barber's shop, at Vera Cruz. Dr. Fenner, from whom I derive the above fact, says: "I myself have seen a man lying quietly in his bed at the Charity Hospital (New Orleans), *reading a book*, while the fatal black vomit was upon him. He seemed to be surprised at the minute inquiries I made about his case, and, as I turned, asked if I thought *he was in any danger*. I gave him an equivocal answer, and left him in blissful ignorance. He resumed his story, and I saw him no more. The next morning he was dead."⁴

An officer said to Dr. Ferguson: "You see I am posting fast to the other world, and you cannot prevent it; but I am as easy as if I was in a post-chaise." Another, the Governor-General of the West India and Leeward

¹ New Orleans Journ., ii. 133.

² Philadelphia Med. and Phys. Journ., xiv. 206.

³ Kelly, Am. Journ., N. S., xiv. 280.

⁴ Fever of New Orleans in 1847, New Orleans Journ., v. 211.

Colonies, “when he contemplated that harbinger of death, the black vomit, pouring from his stomach, on the evening preceding his death, rose from his couch, in full possession of all his acumen, to execute some legal deeds of importance, declaring at the same time, in reply to my dissuasions, he could with equal facility have drawn out a plan for military operations.”¹

In many cases, the ejection of the black matter has been preceded by much nausea and distress at the stomach, as well as by much vomiting. In such instances, the substances thrown up consist usually, at first, of the fluids swallowed, mixed or not with more or less bile. These are followed by mucus containing particles of flocculi, resembling bees’ wings, and soon after by the fully-formed fluid. In some instances, this gradation is not observed, and the patient, after vomiting at first the contents of the stomach, ejects suddenly and unexpectedly a larger or smaller quantity of well-constituted coffee-ground matter. In some few cases, the vomiting is preceded, as Dr. Musgrave has observed, and as I have also seen, by a wedge-like sensation at the cardia, or as if a marble were sticking there. I do not know that this symptom has ever failed to be followed by the ejection in question. But while such is usually the course noticed in respect to the antecedents of the black vomit, it not unfrequently happens that, prior to the discharge of that fluid, the patient had not been troubled with nausea or vomiting; that the stomach had retained everything swallowed—food, drink, or medicine—and that no burning sensation had been complained of before.² In a case which came under my observation in 1853, the patient appeared to be doing well during three or four days. He complained of nothing, was cheerful, asked for food, and relished what was given him. At the end of that time, he suddenly and unexpectedly threw up three or four ounces of a substance presently to be described; shortly after became slightly delirious, soon ejected a small quantity of black vomit, had a convulsion, and died.

The black vomit is sometimes preceded by the ejection of a white, ropy, acid fluid, which makes its appearance at the close of the second stage, and is sometimes discharged in considerable quantities, and with much relief to the symptoms. It is generally attended with considerable retching. This fluid was very frequently noticed during the epidemic of Demerara, in 1837, when it received the appellation of “premonitory” or “precursory” fluid, or “white vomit.”³ In a late epidemic at Barbadoes, referred to by Dr. Davy, the white vomit was not of unfrequent occurrence, though not so common as to arrest attention.⁴ Osgood says that in some cases there is a vomiting, not of black matter, but of a watery substance, of a slimy nature, and with it whatever the patient may have lately taken of drink or medicine (p. 12). In many instances, the substance preceding the black vomit is found copious and colourless (*Ralph*, ii. 66). Cathrall⁵ describes it as of the colour of whey or muddy water, and containing an acid in a free state (p. 9). Dr. Har-

¹ Recollections of a Professional Life, &c., pp. 146, 147.

² Musgrave, Med.-Chir. Trans., ix. 134.

³ Blair, op. cit., p. 80.

⁴ Notes on Blair, ib.

⁵ Mem. on the Analysis of the Black Vomit, p. 4; Fever of 1793, p. 9.

risson states that, in the stomach of an individual whom he dissected at the New Orleans Hospital, he found no black vomit, but a whitish acid-smelling liquid, amounting to about half a pint.¹ Whether these liquids were similar to that described by Dr. Blair, we have no means at present of deciding positively, though we may presume that they differed but slightly from it. In several of the cases of the disease which occurred among us during the two preceding seasons, this white acid matter was ejected. In one instance, when a fair sample of it was obtained, it was thrown up on the fifth day of the attack, and forty-eight hours before the black vomit made its appearance. Dr. Blair found it to occur more frequently than the black vomit itself, in the proportion of 417 of the former to 366 of the latter. The black vomit generally followed the precursory vomit; but many cases of death occurred after the latter only. The earliness of its appearance as antecedent of black vomit is shown by the following table:—

Day of disease	1	2	3	4	5	6	7	8	9	10	11	12	13	Unascertained.
No. of cases in each day	10	66	103	95	72	37	21	8	1	0	1	2	0	1

The period at which this whitish fluid is ejected, its acidity, the probability of its presence in the stomach at the time of the formation of the black vomit, as well as the result of experiments made with it, and to which I shall advert in the next chapter, lead to the supposition that it plays an important part in the formation of that matter.

Except in some cases, when the ejection from the stomach is easily effected from first to last, the expulsive efforts of that organ are of a different kind in the first and last stages of the yellow fever. At first, there are violent retching, straining, suffusion of the face, and spasms of the recti muscles. But when black vomit is established, these violent, distressing, and often painful efforts cease; and, as Dr. Blair remarks, the stomach seems to act alone. It propels its contents by an effort of its own, and apparently independent of extraneous aid, and the matter is, as it were, spouted out, or gulped up rather than regularly vomited—without straining or effort on the part of the patient—almost without his consciousness, and apparently in an involuntary manner; the whole being the result of a jerk or slight convulsive effort, as if effected by a sudden contraction of the stomach, diaphragm, and abdominal muscles; or, it would almost seem, of the stomach alone.² The patient, says Dr. Blair, will speak to you, lean his head over the bed, eject his mouthful, or basinful of black vomit, and resume his conversation with scarcely any interruption. Another writer, Dr. Evans,³ says: “The patient rarely lifts his head off the pillow; but, turning it slightly to one side, by a sudden effort, but without straining, casts a stream from him. The stomach fills again rapidly, and the same phenomena are repeated.”

The first emission of this dreaded fluid frequently takes place suddenly and without warning. It is in such cases unpreceded by nausea or the dis-

¹ *Op. cit.*, p. 148.

² P. 80.

³ P. 248.

tress which usually accompanies vomiting, and brought on by a slight change of posture on the part of the patient, or it follows hiccup, or an effort to discharge flatus from the stomach. The subsequent discharges occur much in the same way; but whether so or not—whether preceded by nausea or otherwise—the emission of the black vomit does not take place in the manner of ordinary emesis. The muscular motion, and the sounds accompanying the ejection are peculiar. There is no violent retching; a loud or hollow sound is heard, caused apparently by a hiccup mingled with a cough. These facts are familiar to all who have witnessed the disease, and have been pointed out by numerous writers.¹

The matter ejected sometimes comes up by mouthfuls, as if in rumination, not unfrequently in a manner which Dr. Daniel, of Savannah, has not improperly likened to that by which a dyspeptic, or a woman far advanced in pregnancy, spits up offending matter.² At other times, perhaps more generally, the fluid is, as it were, pumped up very suddenly, and propelled in a spout, as from an engine, to a very considerable distance. Dr. Rush, in his account of the epidemic of 1793, remarked that the contents of the stomach were sometimes thrown up with a convulsive motion, that propelled them in a stream to a great distance, and in some cases all over the clothes of the bystanders.³ Dr. Harrison has seen it, in the New Orleans Charity Hospital, thrown entirely over the bed of the next patient, and fall on that adjoining;⁴ and similar results have been found to obtain by every one who has observed the disease.⁵

In the yellow fever of this and other cities of the United States, as also in that of Europe and tropical regions, the quantity of the black matter ejected from the stomach, though at first small, becomes soon, in some cases, very large. Dr. Currie, in his account of the fever of 1793 (p. 26), speaks of the quantity thrown up as being enormous. The quantity, says Dr. Cathrall, “sometimes becomes so much augmented, that I have known one gallon vomited in forty-eight hours, besides a considerable quantity, which was of a much thicker consistence, that was discharged by the bowels.”⁶ Dr. Lowber states that, in a case which came under his observation, the stomach contained, upon admeasurement, thirty-two ounces of black vomit.⁷ Dr. Ffirth states that the fluid is thrown up by pints, quarts, and even gallons.⁸ In a case mentioned by Dr. Townsend, of New York, the quantity found in the

¹ R. Armstrong, p. 169; Lewis, *New Orleans Journ.*, iv. 159; Copland, iii. 154, *Am. ed.*; R. Jackson (Spain), pp. 73, 83, 101; R. Jackson, *Sketches*, i. 72; Barry in Boyle, p. 272; Wood, i. 301; Harrison, p. 132; Gros, p. 11; Cartwright, *Med. Recorder*, ix. 10; Imray, *Edinburgh Med. and Surg. Journ.*, pp. 53, 82; Dickson, *Philad. Med. and Surg. Journ.*, iii. 256; Ticknor, *North Amer. Journ.*, iii. 228; Blair, p. 80; Bone, on *Yellow Fever*, p. 3; Finlay, p. 17.

² *Fevers of Savannah*, p. 72.

³ *Works*, iii. 60.

⁴ *New Orleans Journ.*, ii. 132.

⁵ Evans, p. 248; Blair, p. 80.

⁶ *Trans. of Phila. Philos. Soc.*, v. 118; *Ib. Mem.*, p. 5.

⁷ *Med. Museum*, v. 24.

⁸ *A Treatise on Malignant Fever, &c.*, 1804, p. 38.

stomach amounted to two quarts.¹ The disparity between the quantity of the black vomit thrown up and that of the fluids swallowed, is often noticed by the patients themselves. Dr. Bone informs us that an officer, who died of the yellow fever in Barbadoes, in 1821, remarked, that a pint of fluid taken produced a quart of black vomit in no time.² Dr. R. Jackson states that the discharge is "enormously great, so far exceeding the quantity of what has been taken down in drink, as if the stomach had become the outlet of all the fluids contained in the body."³ The quantity of fluid ejected in most cases, says Sir William Pym, who saw the disease in both hemispheres, wonderfully exceeds the quantity drunk; indeed, all the fluids in the body seem to be pouring into the cavity of the stomach; for when it has, to all appearance, been emptied several times, and the patient thinks himself relieved from any painful straining, he is, in the course of a few minutes, without having tasted drink, under the necessity of having recourse to the basin.⁴ Dr. Ticknor speaks of it as astonishingly great;⁵ Gillkrest, as considerable;⁶ Archer, as sometimes incredible;⁷ and Lempriere,⁸ Ralph,⁹ Musgrave,¹⁰ J. Clark,¹¹ A. Hosack,¹² Evans,¹³ Barry,¹⁴ Wood,¹⁵ Harrison,¹⁶ Merrill,¹⁷ R. Arnold,¹⁸ and others, have dwelt with greater or less emphasis on the same fact. The vomiting in all such cases is provoked by swallowing something, either fluid or solid; or by pressure on the epigastrium; but it is often the effect of the mere distension or irritation of the stomach produced by the matter itself; or is excited by a change of position in bed, by sitting, or standing up, and, according to Dr. Blair, by the *act of falling asleep* (p. 80).

When once established, the black vomit very usually continues, without considerable interruption, and is brought up at short intervals, every time nausea and vomiting are excited by any of the aforesaid causes. In some cases, however, especially when it shows itself early, it has been found to intermit, nothing coming up for hours but the medicines and nourishment taken.¹⁹ Dr. Dickson has seen it cease for several days;²⁰ and there are not wanting cases to show that it has occasionally ceased entirely, the stomach retaining everything for some time before death.²¹

The black vomit, notwithstanding its name, is rarely of a black colour. As seen in this city, it is more frequently of a dark-brown, bister, chocolate,

¹ Fever of New York in 1822, p. 191.

³ Sketch, i. 171.

⁵ N. A. Med. and Surg. Journ., iii. 228.

⁷ Med. Recorder, v. 68.

⁹ Med.-Chir. Trans. of Edinb., ii. 67.

¹¹ Fever of Dominica, p. 14.

¹² Hist. of the Yellow Fever of New York in 1795, p. 14.

¹³ Op. cit., p. 248.

¹⁴ Boyle, Med. Hist. Account of the Western Coast of Africa, p. 272.

¹⁵ Pract. of Medicine, i. 301.

¹⁷ New Orleans Journ., viii. 6.

¹⁹ Musgrave, Med.-Chir. Trans., ix. 134.

²¹ Musgrave, loc. cit.

² Essay on Yellow Fever, p. 4.

⁴ Bulam Fever, p. 230.

⁶ Cyclop., ii. 272.

⁸ Diseases of the Army, &c. ii. 65.

¹⁰ Med.-Chir. Trans., ix. 134.

¹⁶ N. O. Journ., ii. 147.

¹⁸ Am. Journ., N. S., iii. 319.

²⁰ Phila. Med. and Phys. Journ., xiv. 206.

or umber hue. In some instances, the colour approaches to a dark slate, or to a muddy claret. It is of two kinds. The one consists of a number of dark flaky particles, which have been not unaptly compared to butterfly or bees' wings, and which assume gradually the appearance, with more or less distinctness, of the grounds of coffee, of soot, or finely powdered charcoal, floating in a quantity, more or less considerable, of thin glairy fluid bearing a slight resemblance to a weak infusion of flaxseed or green tea. The latter fluid, when filtered, differs slightly in colour, being limpid like water, of a deep brandy or rum colour, yellowish or light green. The afore-mentioned flakes or striæ are at first, or throughout in the milder forms of the disease, limited in point of number, and of a light or grayish slate or chocolate tinge. But as the disease advances, and especially in the more malignant cases, they increase in number, and become darker and darker until the whole appears uniformly blackish or even black. The fluid, when completely formed, though homogeneous in appearance when discharged, separates soon, on standing, into two parts; the one consisting of the flaky or coffee-ground matter already mentioned, and the other of the fluid in which they were held in suspension. This flaky matter, which in some cases seems collected in masses of greater extent and entangled in mucus, and at others is divided very minutely and equally mixed, after subsiding to the bottom of the vessel, either in distinct particles or in the form of a dark powder, is readily incorporated with the fluid by the least agitation. When the black vomit is kept a long while, its constituents become perfectly separated, but when shaken and reincorporated, they show less disposition to separate again. In some instances, the quantity of the fluid portion is, from the outset to the close of the attack, very large compared to the solid particles. In others, the reverse is the case; the substance vomited, though presenting all the characteristic features of the coffee-ground or granular matter, being almost or completely deprived of its fluid attendant. In a case I had occasion to see in July, 1853, with Dr. Keating, the black vomit presented that character. In another case, the proportion of solid matter in the fluid thrown up was very small; while, on dissection, sixteen hours after death, the stomach was found to contain about six ounces of thick coffee-ground or granular matter, with scarcely any admixture of fluid.

The other form of the black vomit is more homogeneous in character, and presents the appearance of dark-coloured inspissated mucus or thin tar, or of a thick mixture of molasses and water. In some instances, the matter vomited consists of grumous or dark-coloured blood, fluid or coagulated, without admixture of coffee-ground particles or pale fluid;¹ while in others, again, the matter described above is mixed with coagula of more or less pure blood; or, failing to maintain the character above described, assumes more or less the appearance of blood. In some instances, the discharge towards the termination of the disease becomes nearly sanguineous.

These peculiarities and modifications have been noticed in all our epi-

¹ Cathrall, *Analysis of Black Vomit*, pp. 4, 6, 7; Rush, *Yellow Fever of 1793*, p. 61; Wood's *Practice*, i. 301; Monges, *N. A. Med. and Surg. Journ.*, ii. 57.

demics. They are referred to by Dr. Jackson in his *Account of the Fever of 1820*, p. 81, and not unfrequently presented themselves during the recent outbreak of the disease among us.

Such are the appearances presented by the black vomit in the yellow fever of this city. If we now turn to the descriptions given of it by writers who have observed the disease in other parts of the United States, in Europe, and tropical climates, we shall find them to differ but slightly and on unimportant points from that which precedes.

According to Dr. Hosack, the black vomit, as observed in New York, consists of a dirty-brown water, with a sufficiency of bloody turbid matter, which sometimes falls to the bottom of the vessel, or floats on the surface of the fluid. "This matter, I have observed to exhibit itself in different forms: 1. In flakes like tinder, floating on the fluid discharged. 2. It appears to be discharged in the form of blood and mucus, which gradually subsides. But the more common evacuation is that of the third—the coffee-ground discharge, and which immediately falls to the bottom of the vessel, precisely resembling the appearance of coffee-grounds."¹ At Norfolk, Valentin found the matter vomited to assume a brownish hue similar to coffee-grounds, then to become blackish and of thicker consistence, sometimes resembling tar, or a mixture of soot and water.² Of the characteristic marks of black vomiting, Dr. Smith, of Galveston, says: "They are dark flocculi swimming in a fluid, varying from a brownish, slate-coloured, or whey-looking liquor to one resembling a strong decoction of coffee. In the first portions vomited and in the milder forms, the flocculi are generally few and minute. In more aggravated cases, they are very abundant, and present every variety of shape, like fine powder, and stilliform, linear, or in shreds. In the more malignant cases, the black vomit approaches in appearance to dissolved blood. The flocculi subside very slowly to the bottom of the fluid, and the latter is seen to be of a light-greenish or whey-coloured tinge. Sometimes, however, the flocculent portion swims on the surface, and in appearance is not unlike the inside of the dry mushroom called puff-ball. In portions of black vomit which had been kept a few days, the flocculi which at first had subsided slowly arose and swam on the surface of the fluid. The common comparison of black vomit to a turbid decoction of coffee, probably conveys the best idea of its usual appearance."³ Gros compares it to the lees of ink; Girardin, to India-ink dissolved in water, and keeping in suspension membranous shreds or flakes.⁴ Dr. Middleton Michel, of Charleston, whose microscopical researches on the

¹ Hosack's Practice, p. 389.

² *Traité de la Fièvre Jaune*, 167.

³ A. Smith, *Am. Journ.*, xxv. 503.

⁴ Moultrie, p. 4; Lewis, *N. O. J.*, iv. 159; Dickson, *Am. Journ.*, ii. 72; *Ib.*, *Philad. Med. and Phys. Journ.*, xiv. 215; Nott, *Am. Journ.*, N. S., ix. 281; Simons's Address, p. 30; Davidge, p. 104; Harrison, *N. O. J.*, ii. 132; Kelly, *Am. Journ.*, N. S., xiv. 378; Drysdale, *Med. Mus.*, i. 136; Dalmas, p. 8; Gros, p. 10; Girardin, p. 35; Cartwright, *Recorder*, vol. ix.; Lining, ii. 419; Ticknor, *N. A. J.*, iii. 228; Townsend, pp. 161, 164; C. Drake, *Repos.*, xxi. 135; Seaman, *Webster Col.*, p. 9; E. H. Smith, *ib.*, p. 126; *Fever of N. O. 1839*, p. 331; Shecut, p. 120.

black vomit are entitled to high praise, calls attention to the diversity of colour of the fluid as observed by him. "Though generally reddish-black, it often presented a brown, reddish, or blackish-brown, and sometimes claret colour." The fluid, obtained from a patient who ejected large quantities of it and ultimately recovered, was bright red, recognizable as pure blood with black flocculi in the sediment. In another case, it was of a chocolate colour with a brownish residuum. "Some obtained from my own patients resembled a strong infusion of senna, holding in suspension the ordinary black clots and granules; others, even in a state of rest, were more like a solution of bismuth and India-ink; some again were perfectly black." The intensity of colour, Dr. Michel farther remarks, is much influenced by rest or motion, as it acquires a denser shade whenever the fluid is agitated, and the granules, flocculi, and coagula are made to float; on the contrary, these particles, when the liquid is at rest, settle at the bottom, being specifically heavier than the former, leaving the supernatant liquid comparatively clear, sometimes transparent as serum. "This is not always the case, however, as I have seen the solid portions of the same sample equally divided into such as formed a sediment and such as floated on the surface. The quantity which may be produced and ejected in a given time materially affects the colour, this being of a brighter red whenever the fluid is thrown up in abundance until it frequently amounts to pure blood; whereas, the dark-black and granular aspect with the coffee-grounds sediment, described as characteristic of black vomit, belongs particularly to the smaller portions which are early vomited."¹

In 1828, at Gibraltar, the matter vomited in the last stage of the disease, or found in the stomach, appeared under the following forms; 1. Thin flakes or portions of a brownish-black colour, floating, like broken-up wings of a butterfly, in a glairy fluid or infusion of black tea. 2. A perfect resemblance to a mixture of soot and water, or to the contents of a coffee-pot when the clear part of the coffee has been poured off. 3. A homogeneous, intensely black substance, having a jelly-like consistence, and adhering in great abundance sometimes to the mucous coat.² Pym describes it as a brownish fluid resembling dirty water mixed with a dark-coloured, flaky matter which floats upon the surface, and at last a matter resembling coffee-grounds or thin pitch.³ Others have alluded to its chocolate colour; all have compared it to a mixture of coffee-grounds, in a thin fluid, to the juices of the cuttle-fish, and referred to the flakes or filaments it contains.⁴ Dr. Bone, Deputy Inspector of Hospitals, who, it would appear, paid particular attention to the character of the fluids ejected from the stomachs of persons labouring under

¹ Microscopical Researches on the Black Vomit of Yellow Fever, Charleston Journ., viii. 333.

² Gillkrest, Cycl., ii. 274, note.

³ On the Bulam Fever, p. 230.

⁴ Audouard, p. 60; Pariset, p. 417; Fellowes, p. 54; Caisergues, pp. 169, 170; Berthe, pp. 87, 89; Blin, p. 6; Velasquez, p. 11; Palloni, p. 4; Proudfoot, Edinb. Journ., xxvii. 250; Pym, pp. 35, 41; Boyd in Johnson, p. 300; Rochoux, p. 478; Jourdain, Annales de la Méd. Physiol., v. 260.

the yellow fever, and the results of whose observations on this and other subjects connected with the disease, originally presented in a report dated Barbadoes, 1822, are contained in an inaugural dissertation by his son, speaks of a fluid like Indigo or China ink, brought up with some straining. A brown fluid, resembling urine in appearance; brownish black, not flaky, proceeding from the fauces and gums, and perhaps in some cases from the pulpy cardiac opening of the stomach; brown, flaky blood mixed with mucous matter—a precursor of true black vomit; and lastly, true black vomit, which is also blood altered in its passage through the vessels of the villous coats.¹ Another writer remarks that flocculi of lymph are first seen floating in a straw-coloured fluid. This is followed by the black vomit, “which, on being collected in a basin, appears of a chocolate colour. On allowing it to settle, a sediment resembling the grounds of coffee subsides to the bottom, while the supernatant fluid appears of a straw colour, resembling the serum of the blood. On shaking it together, it reassumes its primary chocolate appearance.”² From all quarters, indeed, we learn that the matter thrown up from the stomach in the latter stage of the yellow fever is characterized as a mixture of fluid more or less limpid with a matter resembling coffee-grounds; that it usually presents dark-coloured flocculi swimming in the fluid; that it assumes at times the appearance of diluted soot; and that, in some cases, it is black as ink or tar; liquid or thick as thin pitch; that in others, it is of a brown-chocolate or a slate colour.³ While such is the usual character of the matter ejected from the stomach, it is sometimes found everywhere, as with us, to consist of blood more or less pure, either red or black, and liquid or coagulated.⁴

Occasionally, the black vomit contains fibrinous masses of greater or less size. In a case of the kind mentioned by Dr. Furlonge, of Antigua, there were two of these masses, one about seven inches long, and the other about four. The larger one looked as if it had been a cast of the duodenum. It may not be unimportant to state, that the patient had been taking, all the previous night, two grains of gallic acid every two hours to check the black vomit.⁵

About the time that the matter ejected from the stomach assumes the afore-

¹ Bone, Essay, p. 3; Gillkrest, op. cit., ii. 274.

² Belcher, Edinb. Journ., xxiii. 250.

³ Holliday, p. 10; Dickinson, pp. 135–8; Desportes, i. 199; Blanc, Dis. of Seamen, p. 437; Hillary, p. 151; Dict. des Sci. Méd., xv. 337; Pugnet, p. 356; Evans, p. 235; Bancroft, p. 12; Blair, p. 81; Madrid, pp. 25–6; McArthur in Johnson, p. 347; Vatable, p. 346; Moseley, p. 438; Savarésy, p. 275; Gillespie, p. 42; Chisholm, i. 174; Jackson, Sketch, i. 72; Osgood, p. 11; Gilbert, p. 66; Caillot, p. 28; Maher, p. 843; Bally, pp. 226, 239; Rochoux, pp. 280, 552; Dariste, p. 162; Imray, liii. 83; Jackson, Tr., p. 261; Pym, p. 230; Wilson, p. 182; Arnold, p. 10; R. Armstrong, p. 169.

⁴ Lining, ii. 419; Pariset, p. 417; Evans, p. 247; Ralph, ii. 68; Jackson, Sketch, i. 3; Dariste, p. 165; Rochoux, pp. 280, 552; Bally, pp. 237, 239; Caillot, p. 19; Maher, p. 842; Archer, v. 67; Blane, p. 437.

⁵ Remarks on the Epidemic Yellow Fever now prevailing at St. John's, Antigua, London Lancet, ii. 440, Am. ed.

said appearances, or a short period before, the intestines give issue to a substance which often bears a strong resemblance, and, occasionally, is perfectly analogous to the former. The passages become brown or black with coffee-grounds, or something not unlike powdered charcoal floating in a serous fluid; or present the characters of molasses, or thin pitch, in regard to smoothness, tenacity, colour, or consistence.

This black inky matter is found on examination to be identical in nature with the true black vomit which, in cases destined to terminate favourably, always comes away by the bowels. It is often intensely acid and acrimonious. I have already referred to this, and cited a case mentioned by Dr. Finlay, in which the matter voided was so acid as to corrode the delft-pan of a closetool, which had never been used before; to such a degree, indeed, that the black marks which were literally branded in it could not be got out, though washed frequently in scalding water. In other cases, the fluid discharged is thin and ichorous, like the washings of flesh. In many, it contains streaks, or a large admixture of blood, and, at times, consists entirely of grumous blood. Intestinal discharges of this kind, bearing a strong analogy to the matter thrown up from the stomach, have been noticed in all places where the yellow fever has appeared.¹ In cases in which these discharges have occurred, as also in those in which they have not been noticed during life, the intestines—sometimes the large, but more generally the small—are found on dissection to contain a more or less considerable portion of the

¹ Moultrie, p. 4; Deveze, p. 24; Ffirth, p. 28; Archer, v. 67; Currie, p. 26; Thomas, p. 84; Drysdale, *Med. Mus.*, i. 136; Stone, vi. 563; Kelly, xiv. 374; Dalmas, p. 9; S. Jackson, pp. 60, 80; Bally, *Typ. d'Am.*, pp. 230, 243, 248; Shecut, p. 120; Gros, p. 11; Girardin, pp. 24, 36, 56; A. Hosack, p. 15; Monson, p. 180; E. H. Smith, *ib.*, pp. 121, 122; Dickson, iii. 256; C. Drake, xxi. 135; Townsend, p. 158; *Ib.*, on Black Vomit, p. 5; Valentin, p. 171; Ticknor, iii. 228; Fenner, *Fev. of N. O.* in 1853, p. 51; Hayne, *Charleston Med. Journ.*, vi. 348; R. Arnold (*Savannah*), p. 318; Bache, *Am. Journ.*, N. S., xxviii. 121; Louis, p. 232; Blin, p. 7; Fellowes, pp. 54, 55; A. Smith, *Am. Journ.*, xxv. 502; Audouard, pp. 58, 61, 186; Caisergues, p. 169; Pym, p. 234; Berthe, p. 86; Gillkrest, *Cycl.*, ii. 271; Boyd (*in Johnson*), p. 300; Velasquez (*in Pariset on Fever of Cadiz*), p. 11; Pariset, *ib.*, p. 30; *Ib.*, *Fever of Barcelona*, pp. 422, 423; *Fever of N. O.* in 1839, p. 333; Warren, pp. 15, 39; R. Jackson, *Sketch*, i. 72, 94; Chisholm, i. 166; Lempriere, ii. 85; McArthur (*in Johnson*), p. 347; Osgood, p. 12; Imray, liii. 81; Bancroft, pp. 10, 12; Hillary, pp. 151, 152; *Madrid*, 2d part, p. 25; Gillespie, p. 42; R. Jackson (*Disease of Jamaica*), p. 262; Pugnet, p. 356; Vatable, p. 346; Desportes, i. 199; Blane on Seamen, p. 440; Belcher, xxiii. 249, 251; Levacher, p. 73; Caillot, pp. 18, 19; Dariste, pp. 163, 165; J. Clark, p. 14; Rochoux, pp. 284, 480; Dickinson, pp. 128, 131; Arnold, p. 10; Evans, p. 257; *Dict. des Sci. Méd.*, xv. 337; Daniel, *Fevers of Savannah*, p. 72; Merrill, *N. O. Journ.*, viii. 6; Dickson, *Am. Journ.*, ii. 73; Hicks, *N. O. Journ.*, v. 221; Slade, *ib.*, i. 86, 94; Hume, p. 200; Musgrave, *Med.-Chirurg. Trans.*, ix. 107; O'Halloran, *Fever of Xeres, &c.*, pp. 82, 127; Chabert, p. 10; Amiel (*in Johnson*), p. 264; Arejula, pp. 172, 424; Waring, p. 51; Grant, p. 33; R. Armstrong, p. 179; Copland, iii. 143, 158; Savarésy, p. 271; Lining, *Ess.*, ii. 423; Burnett, p. 247; Frost, xiii. 32; R. Jackson, *Fever of Spain*, p. 76; Vance (*in Pym*), p. 63; Davidge, p. 103; Gilbert, p. 60; Holliday, p. 10; Simons, *Charleston Journ.*, viii. 368; Paton, of Jamaica, *Lond. Lancet*, Oct. 1853; *Am. Ed.*, p. 288; Blair, p. 94; Chambolle, *Ann. de la Méd. Physiol.*, xiii. 201.

matter.¹ In some instances, it is found in the colon, while the small intestines, and even the stomach, are free from it. As Dr. Nott, of Mobile, well remarks, the matter discharged from, or found in the large intestines, is "frequently dried, resembling pickled walnuts pounded, and sometimes black scybala." Sometimes the black pasty matter is plastered over the whole surface of the small and large intestines, the black colour being more distinct as we approach the stomach. In one case, noticed by Dr. Nott, the whole canal, small and large, was coated with a thick tenacious matter of a purple colour, like blackberry jam, a compound intermediate between blood and black vomit.²

The black vomit has been stated to be odourless.³ Nevertheless, Dr. Blair, while making that statement, remarks that, when distilled, the clear water that comes over has a peculiarly offensive odour, and that, if inspissated, the extract, although not fetid, nor pungent, nor exceedingly disagreeable, produces on some persons instant retching on being smelled. By others, however, a different result has been obtained. Cathrall found the fluid from which the flaky matter had been separated by filtration to have a "faint sweetish animal odour. The matter which remained on the filter had the same odour as the fluid" (pp. 9, 10). Bally speaks of the fetid odour of the black vomit (p. 239). Deveze found it to possess an hepatic smell (p. 24). According to Barry, it had a raw, unpleasant odour, "so peculiar that, on entering the chamber, the state of affairs became immediately manifest."⁴ Dr. Hester says that it has a fresh, disagreeable, nauseous smell.⁵ Dr. F. M. Robertson says the odour is peculiar.⁶ Heastie states that the fluid has a fermentative odour.⁷ Polloni says it was "*materia fetidissima*" (p. 5). Dr. Evans remarks that there is now and then a strong and peculiar odour diffused through the apartment in which this matter is kept, which attaches itself to the furniture and even to the clothing of those who are exposed to it (p. 250). Levacher, who, like Dr. Evans, became familiar with the disease at St. Lucia, makes a similar statement, representing the odour as insipid and ammoniacal, and akin to that of gangrenous eschars, and, in illustration of the power possessed by that odour to attach itself to surrounding objects, cites a striking example of its being perceptible in a bundle of papers which had been left a few hours in a sick room (p. 83). Rochoux found it insipid,

¹ Fellowes, p. 68; Bancroft, p. 24; O'Halloran, p. 189; Deveze, p. 63; Waring, p. 52; Louis, p. 102; Arejula, pp. 418, 421, 422, 426; Audouard, pp. 151, 152; Lowber, Med. Mus., v. 19, 20, 21; Vatable, p. 348; Physick, Med. Rep., v. 130; Ib. in Rush, iii. 92; Pugnet, p. 363; Blane, p. 94; Slade, N. O. Journ., i. 86, 88, 90, 94; Hayne, Charleston Journ., vi. 346 to 628; R. Jackson, Sketch, i. 79, 81, 89, 95; Gillkrest, Cyclop., ii. 276; Gillespie, p. 71; J. Clark, p. 20; Copland, iii. 161; Caldwell, Fever of 1805, p. 99; Savarésy, p. 461; Bally, p. 197; R. Jackson, Fever of Jamaica, p. 265; Ib., Fever of Spain, p. 88; Frost, xiii. 253; Bone, p. 27; A. Smith, Am. Journ., xxv. 505; Kelly, xiv. 378; Rochoux, pp. 360, 361, 535; Dalmas, p. 15; Levacher, p. 76.

² Amer. Journ., N. S., ix. 280.

³ Blair, p. 81; Pariset, p. 419.

⁴ Boyle, Topography and Diseases of Western Coast of Africa, p. 272.

⁵ New Orleans Journ., x. 228.

⁶ Examiner, Oct. 1839.

⁷ Treatise on the Nature and Causes of Yellow Fever, p. 21.

sometimes fetid (p. 552); and Audouard represents it as possessing a putrid smell (p. 7). In the various trials I have made on this subject, the black vomit, when recently thrown up, has usually been found to present the odour ascribed to it by Dr. Hester. In some few instances, the smell was very disagreeable, and almost fetid.

The ejection of the black vomit is generally preceded and accompanied by a sensation of soreness and burning in the stomach, along the œsophagus, and in the fauces and mouth; as well as by anxiety and pain at the præcordia.¹

From this and other circumstances, the matter of the black vomit has been supposed to be of an acrid and excoriating or even corrosive nature.² But, although such may often appear to be the case, owing to the peculiar composition of the fluid, the state of the secretions, and the morbid sensibility and inflamed condition of the parts over which it passes, there is little doubt that, except under certain circumstances, the fluid is void of the excessive acridity ascribed to it.³ In the experiments of Cathrall, it imparted, it is true, an acrid sensation when applied to the lips. But on the tongue it did not produce the least corrosive effect. Nor did it affect the skin of the hands and different parts of the body. Cats, dogs, and fowls were fed with it without any injurious, or, indeed, sensible effects on their digestive organs; while fumes, obtained by the evaporation of the matter, proved inoffensive to those who inhaled them.⁴ Like the last-mentioned writer, Dr. Ffirth fed dogs and cats with the matter of the black vomit during days and weeks; he did more—he inoculated dogs with the fresh matter, subjected himself to the same operation, applied the fluid to the surface of a cut made on his arm, secured it there for two days by means of sticking-plaster, and repeated the experiment above twenty times in various parts of his body. He inserted the matter in his eyes—swallowed a large quantity diluted, or pure; but from these experiments no injurious effects ensued. When the matter was applied to wounds, very slight inflammation sometimes, though not always, resulted, and the wounds healed kindly. In general, not more inflammation followed “than would have occurred had no black vomit been introduced.” When it was dropped into the eye, “it felt a little uneasy for about a minute; but produced no pain or inflammation. I have,” says Dr. F., “frequently had cold water to produce the same effect.” When swallowed, it produced no

¹ Rush, iii. 60; Kelly, p. 377; Evans, pp. 233, 256; Fellowes, p. 54; R. Jackson, i. 68, 72; Barton, p. 10; Waring, p. 45; Blane, p. 438; E. H. Smith, p. 118; Bally, p. 216; Dickinson, p. 131; Girardin, p. 171; Campbell, of Charleston (in Watts), p. 250; Boyle, p. 203; Pym, pp. 228, 229; Ticknor, North Amer. Journ., iii. 222; Rochoux, pp. 289, 480; Grant, p. 31; Lewis, N. O. Journ., iv. 159; Musgrave, Med.-Chirurg. Trans., ix. 134; Bryson, p. 66; Nott, Am. Journ., ix. 282.

² Th. Cooper, Discourse on the Connection between Chemistry and Medicine, Philad. 1818, p. 34; Dariste, p. 160; Dalmas, p. 8; Valentin, p. 166; Desportes, i. 203; Pugnoet, p. 356; Gros, p. 10; Rochoux, p. 290; Girardin, p. 33; Diet. des Sci. Méd., xv. 336; Nott, Amer. Journ., N. S., ix. 282; Lefoulon, p. 49; Audouard, p. 6.

³ Physick, Repository, v. 130.

⁴ Cathrall, pp. 20, 21, 22, 23.

nausea or pain—no more effect, indeed, than if so much water had been taken.¹ Dr. Chervin, who on several occasions swallowed a quantity of the matter of black vomit, felt no inconvenience from it.² Neither did Dr. Guyon, in the numerous experiments he made at Martinique in 1822.³ Dr. McKinnal, of the British navy, for certain reasons which need not be stated here, swallowed a wineglassful of fresh black matter, and felt no inconvenience therefrom. “It did not impair his appetite for dinner.”⁴

To the taste, the black vomit is at times more or less insipid.⁵ In some instances it has been found to have a saline and bitter taste.⁶ More frequently, however, it possesses an acid flavour, of which patients often complain, and which gives rise to the sensation of acridity, and to the excoriating effects just referred to. It is acid in reaction. Litmus paper is turned red by immersion in it, and turmeric paper, changed by an alkali, is restored to its original colour. It effervesces—not, perhaps, with the earth on which it falls, as Hippocrates said of the black matter thrown up in some cases of the fever he describes, but with alkaline carbonates, requiring sometimes a large quantity of potash to neutralize it;⁷ and the presence of a predominant acid, the hydrochloric, is proved by the effect upon it of the nitrate of silver, which throws down a white precipitate, which is again redissolved by ammonia, but not by nitric acid.

Observations made in Jamaica showed the existence of an oily fluid floating on the surface of the black vomit in sporadic cases of yellow fever, which there is common; and that, too, in instances where no oleaginous medicines had been administered.⁸ From experiments, long ago made at Barcelona, and the results of which were confirmed by a competent chemist of Paris,⁹ the presence of this oleaginous fluid would seem to have been ascertained. It was there found that the quantity of this substance was increased by the addition of acids.¹⁰ I am not aware that anything of the kind has been dis-

¹ A Treatise on Malignant Fever, pp. 53, 54, 55, 56, 57. Phila. 1804.

² Rapport lu à l'Acad. R. de Méd., p. 30.

³ Lefort, Mém. sur la non Cont. de la F. J., pp. 31, 126.

⁴ Bryson, op. cit., p. 55.

⁵ Physick, Med. Reposit., v. 129; Cathrall, Phila. Trans., v. 128; Gros, p. 9; Evans, p. 224; Wilson, p. 182; Arnold, p. 9.

⁶ New Orleans Journ., x. 228.

⁷ Nott, Am. Journ., ix. 281; Monges, N. A. Journ., ii. 57; Lyons, Lond. Med. and Phys. Journ., 1828; Lewis, N. O. Journ., iv. 159; F. M. Robertson, Examiner, Oct. 1839, ii. 665; Harrison, p. 148; Evans, p. 249; Blair, pp. 80, 81; Cathrall, p. 11; Davy, Fever of Barbadoes, Edinb. Journ., lxxii. 280; Ib., notes to Blair, p. 81; Riddell, N. O. Journ., ix. 420; Michel, Charleston Journ., viii. 335; Finlay, p. 17.

If liquor potassæ be added to black vomit beyond what is required for neutralizing the acid, the granular or flocculent sediment becomes dissolved, and the vomit becomes homogeneous and perfectly clear, like light-coloured port wine. In one case, \mathfrak{z} i of black vomit, mixed with an equal quantity of water, required \mathfrak{z} ii of liq. potass. to render it transparent and homogeneous.—Blair, p. 81.

⁸ Davy's Notes on Blair, p. 81.

⁹ Laugier, Professor at the Museum of Nat. Hist.

¹⁰ Pariset, p. 636.

covered in this city or in any part of this country where the black vomit has been carefully examined. In some instances, though not in all, the microscope certainly revealed the presence of a notable quantity of oil-globules; but as most of the individuals who had ejected the matter experimented upon, or in whose stomachs it was found, had taken spirits of turpentine, castor oil, or essence of beef, there is reason to believe that the globules in question were derived from these substances, and constituted no essential part of the fluid containing them.

Much has been said in olden and modern times respecting the source, nature, and manner of production of the black vomit; for, although we occasionally meet with authors who, like Dr. Daniel, of Savannah,¹ frankly avow that, from all they have observed, the only conclusion to which they can satisfactorily arrive, concerning those several points is, that they are yet to be explained; the very large majority of those who have written on the yellow fever, feeling less discouraged, have endeavoured to reach definite conclusions on the subjects in question, and enforced their views with more or less prolixity and pertinacity. Early, however, as attention has been called to the source and nature of the black vomit, and zealously as the investigation has been conducted, considerable difference of opinion existed in former days, and, indeed, continued until recently to exist, on the subject. On few questions, indeed, have more discordant theories been advocated.

It will not be necessary to examine in detail all these theories; for, independent of the circumstance that such a survey would require more space and time than the nature of the present work will justify, not a few of them have been so ephemeral in their character, and are so evidently destitute of foundation that they merit but slight, if any, attention. In the present state of our knowledge respecting the pathology of the yellow fever, it would be a waste of time, for example, to inquire into the merits of the opinion entertained formerly by a few writers, that the black vomit is exclusively or principally the result of a gangrenous or sphacelated slough or exfoliation of the coats of the stomach macerating in the secreted fluids of that organ. Few pathologists of the present day admit the existence of such a condition in yellow fever. Even if real sphacelus or gangrene of the stomach were to occur, it is doubtful whether it would give rise to a fluid like the black vomit, in such large quantities especially. The fluid, besides, is ejected in cases in which recovery takes place, and in which, therefore, there cannot be a shadow of probability of gangrene having occurred; while it has been found, on dissection, in stomachs the soundness of which could not be disputed. Still less need we dwell on the idea thrown out by Fordyce,² and subsequently adopted by Breschet and Heusinger,³ that it is an exudation similar to that producing the incrustation upon the tongue, lips, and teeth, in malignant fever. The former has no advocates at the present day, and the second has

¹ Autumnal Fevers of Savannah, p. 72.

² Dissertations on Fevers, p. 335, Am. ed.

³ Researches on the Accidental Production of Pigments and Carbon in the Human Body.—See *Cyclop. of Pract. Med.*, iii. 94.

died with its celebrated promulgator and his few followers. More useful will it be to examine those theories which have commanded the attention of the profession at large, and continue to this date to enumerate advocates.

By many experienced and accurate observers, the black vomit was, and by some contemporary pathologists is, regarded as consisting of little more than altered bile, sometimes, though not necessarily, mixed with blood.¹ This opinion is principally founded on the supposed identity of the yellow fever with the remittent bilious fever, in which the vomiting of bile of various degrees of purity is a very ordinary, if not universal symptom; as well as on the circumstance that bile often assumes a dark or even black colour; that a fluid more or less allied in appearance to the black vomit has been discovered not only in the stomach, but in the common duct and duodenum; that in some cases of yellow fever a fluid, the bilious character of which is undisputed, is ejected from the stomach; that the real black vomit occurs often in cases where there has been no effusion of pure blood, at least in which none has been observed during life or discovered after death; that the black vomit bears no resemblance to the latter fluid, and that it is almost invariably associated with jaundice.

Others have regarded it as of hepatic origin, but not as of a bilious nature—the product of a perverted or vitiated secretion of the liver. Such was the opinion especially of the late Dr. Cathrall, of this city, whose experiments on the black vomit attracted some attention at the period of their publication.² Disbelieving the opinion of its being putrid blood, because blood after becoming highly putrid and kept for six months imparts a red colour to water, a property not destroyed by a high degree of putrefaction; arguing also that blood differs from the black vomit in not consisting of flaky particles, and giving no proof of containing an acid in a disengaged state; opposed besides to the belief of its being bile, either putrid or rendered black by admixture in the stomach with nitric acid, which was supposed by Dr. Mitchell, of New York, to be generated in that organ and the intestines, and at the same time believing that dissection had shown the black flaky particles or colouring matter of the vomit to proceed from the gall-bladder, Dr. Cathrall thought himself justified in referring it to the cause mentioned. In illustration of the correctness of this opinion, he called attention to the circumstance proved by many facts, that the secretory economy of the liver may be so far arrested in its healthy action, by the progress of disease, as to assimilate a fluid having not the least analogy to bile, in the same way that the kidneys secrete sometimes a fluid not at all like urine.

¹ Desportes, i. 202; Towne, p. 21; Lining, *Edinb. Essays and Obs. Med. and Phys.*, ii. 419; Deveze, p. 24; Drysdale, *Med. Mus.*, i. 136; Davidge, *Notes to Am. ed. of Bancroft*, p. 506; J. Clark, *Fever of Dominica*, pp. 13, 66; Berthe, p. 87; Robert, *Guide Sanitaire*, &c., i. 337; A. Hosack, *Yellow Fever of New York in 1795*, p. 14, note; Stuart, *Diss. on the Salut. Effects of Mercury*, &c., *Caldwell's Select Med. Theses*, 1806, p. 48; J. Mitchell, *Med. and Phil. Register*, iv. 185; Heastie (A.), *Yellow Fever*, p. 22; McCabe, *Diseases of the West Indies*, p. 24.

² *Transactions of the American Philosophical Society*, v. 125, and *Memoir on the Analysis of the Black Vomit*. Philad. 1800.

By others, again, the black vomit has been viewed as the product of a morbid secretion of the inflamed vessels of the stomach, which, in the healthy state, secrete mucus and the gastric fluid. Of the advocates of this opinion, the greater number regard this secreted fluid as being of a sanguineous nature. Such was the opinion entertained on the subject by the late Dr. Physick, whose experiments on the nature of the fluid were made during one of our early epidemics.¹ Such also was the sentiment, more or less modified, of Drs. Rush,² Caldwell,³ Stuart,⁴ Ffirth,⁵ Lowber,⁶ and Sanderson,⁷ of this city; of Drs. Shecut⁸ and Dickson,⁹ of Charleston; Belcher,¹⁰ H. M'Lean,¹¹ R. Jackson,¹² Dancer,¹³ and J. Wilson,¹⁴ of England. By the last-mentioned writer, whose views on all subjects connected with the etiology and pathology of the yellow fever are entitled to respectful consideration, the substance vomited is maintained to arise from the "perverted chemical action of the secretions of the stomach, intestines, and liver, especially of the first," and to be neither vitiated bile nor extravasated blood, since it possesses not the properties of either. "It appears," says Dr. Wilson, "that nature, in her distress, to relieve the turgid vessels of those viscera, pours forth the redundant blood through secretory vessels by which it is changed." Dr. M'Lean remarks that, "from the great determination of blood and the violence of the action in the vessels, immense secretions are made in the stomach, which, acquiring there a dark colour from the admixture of other fluids, and perhaps portions of the coats of the stomach," with some blood and bile, create what is termed the black vomiting.

Dr. R. Jackson, who originally thought that the black colour of the matter ejected arose from admixture of the black bile of the gall-bladder with the fluids of the stomach, subsequently came to the conclusion, from the results of observations on the dead body, that the colour proceeded from admixture with diseased secretions from the mucous membranes of the whole gastric system, more especially of the liver, and discharged from ducts or canals, not bloodvessels, the mouths of which, filled with dark-coloured fluid, he discovered in various parts of the viscera. More recently, Dr. Dickson, in the publication referred to, asks the question, Is it (the black vomit) or is it not a mixture of dark blood, acted upon and closely combined with the gastric fluid? and answers it in the negative, "because, in the vast majority of instances, it is impossible to detect any of the reddish tint which blood, however altered, essentially possesses and retains." This tint it communicates in all cases of hæmatemesis proper to the contents of the stomach, so as to be unequivocally perceptible. In many patients, in the last stages of yellow

¹ Medical Repository, v. 129.

³ Fever of 1805, pp. 91, 92.

⁵ Dissertation on Yellow Fever, p. 37.

⁷ Ibid., v. 20.

⁹ Phila. Med. and Phys. Journ., xiv. 229.

¹¹ Mortality of Troops in St. Domingo, pp. 29, 30, 89.

¹² Sketch, &c., i. 80, note.

¹⁴ On West Indian Fever, p. 211.

² Works, iii. 60, 61.

⁴ Essay on Mercury, p. 48.

⁶ Med. Museum, v. 26.

⁸ Med. and Ph. Essays, p. 120.

¹⁰ Edinb. Med. Journ., xxiii. 252.

¹³ Med. Assistant, p. 83, note.

fever, and occasionally of bilious fever, this hemorrhage occurs. It is then easy to discover the mixture of blood with black vomit in other matters ejected. A bit of white paper or rag dipped in any such mixture, in which the smallest proportion of blood is diffused, will not fail to show plainly enough the reddish hue.

From what precedes, as well as from what we gather in the writings of those who advocate the secretory nature of the black vomit, it will be perceived that the theory is little better than a conjecture. But it was the best that could be adopted, in the absence of information necessary to arrive at something more satisfactory, to account for the formation of the fluid. Its advocates were convinced, from the numerous facts observed, and a variety of circumstances to which attention will be called presently, that the black vomit differs essentially from bile or the product of any morbid operation of the liver. They perceived very plainly that it is the product of a diseased condition of the mucous surface of the stomach, probably also of the intestines, and that it is not poured into those organs from any neighbouring or distant part. They entertained, besides, the belief that, though issuing from the minute bloodvessels of the stomach and bowels, which are sometimes found gorged with it, and the inflamed or congested state of which is evidently relieved by its effusion, the matter in question nevertheless differs too much from ordinary blood to justify its being regarded as simply a modified or altered condition of that fluid, produced after its discharge into those cavities, or just before, through the agency of their morbid products. And having satisfied themselves on those points, but being at the same time unaware of many facts which have more recently come to light, and to which I shall soon advert, the pathologists mentioned could not well have avoided attributing the change effected in the blood to a vital process analogous to that which takes place in secretion. How far this opinion is entitled to our regard will be seen as we proceed.

By another and much more numerous set of writers the black vomit is viewed in the light of an hemorrhagic effusion from the capillary vessels of the mucous coat of the stomach and bowels; in other words, of blood—real, though somewhat modified in its texture. This theory of the nature and source of this important fluid, though claimed as new by physicians of our day, may be traced up to an early period in the history of the yellow fever. It was advocated, in the plainest possible terms, more than a century ago, by Dr. Henry Warren, of Barbadoes, whose letter to Dr. Mead, on the epidemic which prevailed in that island in 1732, was issued in volume form in 1740, and constitutes one of the earliest professional records of the yellow fever in our language. “I ought to observe here,” says Dr. Warren, “that the fatal black stools and vomitings are vulgarly supposed to be only large quantities of black bile or choler; which false notion seems to be owing to that fixed, unhappy prejudice that the fever is purely *bilious*. But, let any one only dip in a bit of white linen cloth, he will soon be undeceived, and convinced that scarce anything but mortified blood is then voided; for the cloth will appear

tinged with a deep bloody red or purple, of which I have made many experiments."¹

Closely allied to these views, relative to the nature of the black vomit, are those of Dr. John Williams, who, in an essay on the yellow fever of Jamaica, which appeared in 1750, enumerates, among the worst symptoms of the disease, "strong and continual convulsions of the diaphragma, intercostal muscles, and stomach; æruginose vomitings, then vomiting of black adust blood, appearing like the grounds of coffee mixed with acrid unfinished bile; the juices of the stomach and pancreas both very sharp."² A similar opinion was about the same time adopted by Dr. Chevalier in his letters on the diseases of St. Domingo (p. 10), and other of the older writers, and has since been advocated by the highest professional authorities in this and other countries,³ some of whom had entertained at one time a different sentiment on the subject.⁴

¹ A Treatise concerning the Malignant Fever in Barbadoes and the Neighbouring Islands, &c., pp. 39, 40.

² Essay on the Bilious or Yellow Fever of Jamaica, 1750, p. 16.

³ Lind, *Hot Climates*, p. 185; Bruce, in ditto, p. 186, note; Rouppe, *Diseases of Seamen*, p. 304; J. Hunter, p. 64; Blane on *Seamen*, p. 410; Hillary, p. 151; Pugnet, pp. 356, 357; Lempriere, ii. 103; Stevens on the *Blood*, p. 330; Bancroft, pp. 39-43; Bone, in *Cyclop. Pract. Med.*, ii. 274; Gillkrest, *ib.*, p. 274; Rochoux, pp. 279, 552; Imray, *Edinb. Journ.*, liii. 91; Vatable, *Ann. Mar.*, for 1828, p. 355; McArthur (in Johnson) on *Trop. Clim.*, p. 350; Monges, *N. A. Med. and Surg. Journ.*, ii. 58, 59; Parrish, *Med. Museum*, iii. 189; Valentin, p. 168; Dubrueil, *Journ. Univ.*, viii. 321, 322; Dariste, p. 116; E. H. Smith, in Webster's Collection, pp. 120, 121; Maher, p. 893; Guyon, *Fever of Gibraltar*; *Ib.*, for 1830, p. 755; Lyon, *Lond. Med. and Phys.* for 1828; Arnold, p. 38; Pariset, *Fever of Barcelona*, 1821, pp. 361, 389, 638; Kelly, xiv. 382-385; Waring, p. 52; Osgood, p. 26; Pym, p. 223; Evans, p. 248; Barry, in Boyle, p. 272; Chambolle, *Broussais's Journ.*, xiii. 200; Furlong, xxv. 291; Blair, p. 81; J. Davy's notes on Blair, p. 81, and *Edinb. Med. and Surg. Journ.*, lxxii.; Nott, ix. 281; Harrison, ii. 119, 147; Audouard, *Recueil de Méd.*, p. 1, and F. J. de Barcelone, pp. 188, 189; Levacher, p. 83; Palloni, p. 5; Townsend, 1822, p. 162; S. Jackson, p. 81, and *Phila. Med. and Phys. Journ.*, ii. 22; Irvine, pp. 24-26; Louis, p. 218; Desmoulin, *Eccl. Repert.*, xiii. 164; Ashbel Smith, xxv. 503; *Ib.*, *Trans. of N. Y. Acad. of Med.*, i. 61; Fenner, *Southern Med. Rep.*, i. 581; *Ib.*, ii. 89; *Ib.*, *Fev. of 1853*, p. 50; Lewis, *New Orleans Journ.*, iv. 159; Dickinson, pp. 134, 135; Ferguson, *Recollections, &c.*, p. 146; Chervin, in Waring, p. 50; Merrill, *N. O. Journ.*, viii. 6; Grant, pp. 32, 47; *Edinb. Med. and Surg. Journ.*, lxix. 124; Michel, *Charleston Journ.*, v. 748, 749; *Ib.*, viii. 333, 348; Shannon, *Pract. Ob.*, p. 48; Craigie's *Practice*, i. 247; Copland, i. 979; *Ib.*, iii. 145; Dickson, *Eccl. Journ.*, iv. 110; Carswell, *Cyclop.*, iii. 88, 102.

⁴ M. Audouard, whose earliest publication on the yellow fever appeared some eighty years after that of Warren, strangely enough sets himself up as having been the first to discover that the black vomit is not bile, but simply the result of an hemorrhagic effusion. He rates his fellow-commissioner, M. Pariset, very roundly, for having appropriated to himself what he holds in the light of his discovery, and entered into, without acknowledgment, all the particulars he had laid before the public two years before—even to the tasting of the black vomit (p. 6, note).

It would be easy to show that, in everything sensible M. Audouard has said on the subject of the black vomit, or indeed of anything connected with the yellow fever, he has been anticipated. Of his publications we may say what Blumenbach is reported to have remarked of Gall's phrenological works: "Everything good they contain belongs to some one else, and every novelty they teach is good for nothing."

CHAPTER XIII.

BLACK VOMIT CONTINUED.

FROM an attentive survey of all the circumstances connected with the discharge of the matter of the black vomit; of the symptoms and pathology of the diseases in which it is observed; of the anatomical lesions revealed on dissection, and of other characteristic peculiarities, I am led to coincide with those who advocate the last of the opinions mentioned in the last chapter. It derives countenance from a variety of facts and circumstances of both a negative and positive character. 1. The impossibility, in the present state of our knowledge, and on a full consideration of the characters of the fluid, of adopting either of the other theories stated. 2. The result obtained from a careful examination of the several materials which enter into the composition of that fluid.

The fallacy of the ancient belief that black vomit is vitiated black bile, was pointed out as far back as the days of Dr. Henry Warren,¹ of Barbadoes, to whose work I have already referred, and has been demonstrated in the most positive manner by several of our immediate predecessors and contemporaries, Physick,² Bancroft,³ Ffirth,⁴ Monges,⁵ Lowber,⁶ Robertson,⁷ Dickson,⁸ and Arnold.⁹ The theory is disproved on the following grounds:—

1. The yellow fever is not a disease of a bilious character. In it, as a general rule, the secretion of bile, so far from being increased, is greatly restrained. Indeed, it often happens that, after a day or two, the formation of the fluid is nearly, if not completely suppressed; and while the absence of bile in the evacuations is invariably to be viewed as a sign of dangerous import, the reappearance of the fluid is always hailed as an indication of amendment. Such being the case, it would be impossible, supposing the black vomit to be bile, to account for its proving so generally the forerunner of a fatal issue.

2. The quantity of black vomit discharged is generally too large at each act of ejection, and then too frequently repeated, to justify the belief that the matter proceeds from the liver or gall-bladder.

3. The true black vomit is often found to be voided in large quantity; and the stomach, after death, is discovered to contain much more in cases in which

¹ A Treatise concerning the Malignant Fever in Barbadoes, p. 39.

² Med. Repos., v. 129.

³ An Essay on the Disease called Yellow Fever, p. 27.

⁴ A Treatise on Malignant Fever, p. 37.

⁵ North Am. Med. and Surg. Journ., ii. 58, 59.

⁶ Med. Mus., v. 26.

⁷ Med. Examiner, Oct. 1839.

⁸ Essays, i. 356.

⁹ A Practical Treatise on the Bilious Remit. Fever, p. 38.

the liver is found either healthy or in a condition precluding the possibility of its secreting bile or any fluid approximating to it. The idea of an apparently *healthy* organ secreting immense quantities of a most *unhealthy* substance, is scarcely to be entertained. The same may be said of the supposition of an organ, exsanguine, pale, and brittle, more or less filled with oily matter, often in a condition very similar to that of a common fatty liver, exhibiting little or no appearance of containing bile on being sliced, as is most generally the case in the yellow fever, pouring out pints or gallons of such a fluid.

4. The stomach, or intestines, or both, have been found at times filled with this black matter, while in the same subject the bile in the gall-bladder, biliary ducts, or the *pori biliari*, was in its natural state, or presented no analogy, as regards colour and appearance, to the former fluid. Dr. Physick has found the gall-bladder filled with a fluid of a brickdust colour, or a light green, or transparent and colourless or purulent colour. Dr. Lowber informs us that he saw more than thirty ounces of black vomit in the stomach of a subject whose gall-bladder was small and contracted, and contained a very inconsiderable portion of viscid bile—results which could not obtain were black vomit the product of the liver.

5. The pylorus, in several instances, has been found closely contracted, and yet the stomach contained black matter.

6. In external appearance, the matter of the black vomit differs greatly from dark coloured or black bile. The latter is of an homogeneous nature, smooth and glossy, and, when examined closely, is found to be of a really black or deep green colour; whereas the former is, as already stated, in the majority of cases, a compound of flaky or granular particles, of a dark brown rather than black colour, floating in a thin, pale, or reddish fluid. Even when the black vomit is of a thick and tarry appearance, it may be distinguished from bile by other signs.

7. When mixed with water, the two substances produce dissimilar effects. The bile unites readily with the water, imparting to it a greenish tinge; while the grounds of the black vomit float on the surface of the liquid in the same manner as bran deprived of its mucilage, or rather as mahogany sawdust.

8. If the darkest coloured bile be spread thinly over a white surface, such as the skin, it loses the colour it had in its accumulated state, and appears of a yellowish green colour; but if the black vomit is treated in the same way, it retains its black or dark brown appearance.

9. When a bit of paper or white muslin is dipped into these two substances, it is very differently coloured by them. The bile imparts a greenish, the black vomit a reddish tinge to it. We have seen that this fact was long ago pointed out by Warren. Since his time, it has been referred to by Monges, Robertson, and others.

10. When bile is added to black vomit, it alters its nature so much as to give it an appearance different from that which it had before.

11. The black vomit differs very much from any mixture that can be made of dark coloured bile with any of the fluids found in the stomach or intestines.

12. The bile and black vomit differ as to taste. The former always im-

parts a bitter taste, of greater or less intensity; the other is sometimes insipid, more frequently acid.

13. The black vomit is sometimes found to adhere to and line those portions of the mucous membrane that are inflamed or congested, leaving the other and healthy parts uncovered. In some cases, none of the fluid is found loose in that organ, though a large portion of the internal surface is thickly smeared over in that way. When scraped off, it cannot be made to adhere again in the same manner; from which we may infer that it has exuded from the vessels to which it is attached, and has not been poured into the stomach from some other organ. Bile is never found to adhere in this way to the lining membrane of the stomach, and especially to select in preference its inflamed portions. It tinges, and mixes with, the mucus secreted by these parts.

14. Although in cases in which black vomit has been discharged freely, or in which, though not ejected, it has been found after death, dissection reveals an engorged state of the capillary vessels;¹ yet in very many, probably the majority of instances, the engorgement of the vessels appears to be lessened or removed by the formation and ejection of the matter. Such was the opinion, long ago expressed, of Dr. Physick.

“The secretion of black vomit,” as is remarked by that eminent surgeon, “appears to be one of the most common modes in which violent inflammation of the stomach has a disposition to terminate. Death, however, in general, takes place before it entirely disappears. I have seen many cases which show that the inflammation is diminished by the secretion, of which it will be sufficient to mention the following: On opening a stomach, one-half of it was coated with adhering black matter, while the other half was free from it; on scraping it off clean, and comparing the part underneath with the other half of the stomach which had not secreted any black matter, the difference in the degree of inflammation was very striking, being much the least in the part which had been covered with the black substance. In some cases, where the vomiting of black matter had been considerable in quantity, or continued for several days, the inflammation was found very faint indeed.”²

This tendency of black vomit to relieve the engorgement of the capillaries has been noticed by very many other writers.³ Need it be remarked that the effusion of bile—its ejection from, or its accumulation in, the stomach—has never produced any effect of the kind?

15. Facts innumerable may be adduced to show that the black vomit is formed in the stomach itself, and is not poured in that viscus from the liver or any other organ. “The colouring matter may be, and often is,” as Dr. Dickson remarks, “seen in the vessels of the stomach itself, from the mouths

¹ Harrison, N. O. Journ., ii. 139; Hayne, Charleston Journ., vi. 628; Ffirth, p. 36; S. Jackson, p. 78.

² Med. Repos., v. 131, 132.

³ Rush, Fever of 1793, p. 94; *Ib.*, Works, iv. 44; Townsend, p. 191; Copland, iii. 143, 161; Parrish, Med. Museum, iii. 168, 188, 190; Lowber, *ib.*, v. 19; Blair, p. 92; Harrison, N. O. Journ., ii. 139, note; Rochoux, p. 355; Waring, p. 52; S. Jackson, p. 78; Blair, p. 92; Fenner, *Fev. of N. O. in 1853*, p. 50; A. Smith, xxv. 504, 505; *Ib.*, Trans. N. Y. Acad. of Med., i. 64.

of which it may be pressed without difficulty by the finger.”¹ The same fact has been recorded by Dr. Arnold, of Savannah.² Dr. Evans relates a case in which a large quantity of black vomit was ejected, and a great deal found in the stomach after death. “Its mucous coat [of the stomach], washed and examined with care, was of a gray colour throughout, except that it was speckled with innumerable black spots, produced by this black matter, stopping up the mouths of the exhalant vessels, and easily picked out with the point of a needle”³ Analogous statements are made by Ffirth (p. 37), Kelly (*Am. Journ.*, N. S., xiv. 382), Arnold, of Jamaica (p. 38), Gillkrest (*Cyclop.*, ii. 274, 275), R. Armstrong (p. 178), Ashbel Smith (xxv. 505). “The veins of the stomach,” says Dr. R. Jackson, “were generally turgid; the villous coat was abundant at some places, loose and in the act of separating at most; the surface underneath the separated villi was streaked with bright or dark red, or was studded with clusters of points not unlike measles—most numerous at the upper orifice, but not confined to it. In some instances, the mouths of canals were visible at different points of the interior surface, yielding a dark-coloured fluid by pressure” (i. 79).⁴ Dr. Physick, long ago, pointed out the fact that cases occur in which the inner coat of the stomach is as black as the black vomit, and that, in many such cases, no black matter is found in that viscus; an effect evidently due to the retention of the matter in the vessels, inasmuch as the dark colour of the membrane is lessened when the matter is poured out in the stomach.⁵

16. And, finally, the black matter, or one similar to it in its physical characters, and doubtless in nature, has been found at times to be formed in, or to proceed from, parts where bile in substance cannot, by any possibility, penetrate, and whence it cannot be ejected. It has not unfrequently happened, for example, that the application of a blister, especially in the advanced stage of the disease, has been followed by a copious exudation of fluid resembling in all respects the matter ejected from the stomach. Dr. Monges, a competent

¹ *Med. and Phys. Journ.*, N. S., v. 209; *Essays*, p. 356.

² *Am. Journ.*, N. S., iv. 318.

³ Pages 33, 216, 249–275.

⁴ The yellow fever is not the only disease in which the above phenomenon presents itself. It occurs sometimes in fatal cases of hæmatemesis, or in complaints which have been attended with discharges of blood by the stomach or bowels, or both. Portal, in his essay on *Melæna*, published originally in the *Memoirs of the Medical Society of Emulation*, of Paris, and since inserted in the second volume of the collection of his *Essays*, relates several highly interesting cases of this disorder, in which the patients vomited and voided per anum a greater or less quantity of black fuliginous fluid. This the able and celebrated physician ascertained to be not bilious or atrabilious matter, but altered blood. On dissection of three of these cases, the fluid could be easily forced out of the engorged capillaries. It may not be uninteresting to state that one of these individuals was no less a personage than the Comte de Vergennes, Minister of Foreign Affairs under Louis XVI., and to whom this country was so much indebted during our revolutionary struggle.—*Mémoires sur Plusieurs Maladies*, ii. 138, 163, 170, 208.

⁵ *Loc. cit.*, p. 131.

authority in such matters, states his having seen this in 1820.¹ A similar fluid has often been found in the bladder.²

In a case which occurred in the Pennsylvania Hospital, in September, 1854, the vessels under the mucous membrane of the bladder were gorged with a matter of a greenish bistre colour, which, on examination under the microscope, was found to present the characters of the black vomit. The urine contained the same matter. For this fact I am indebted to my friend, Dr. James Darrach, house surgeon to the hospital.

The fluid has been found exuding from the nostrils, fauces, tongue, eyes, and ears.³ The vessels of the œsophagus have been found gorged with it. A fluid analogous to it has also been detected in the pericardium.⁴ It has been discovered in the pleural cavity.⁵ Dr. Merrill mentions its having been seen in the peritoneum. The following history of a case which occurred in the practice of the late Dr. Monges, during the epidemic of 1820 in this city, and which I myself witnessed, presents an apt and striking illustration of the last-mentioned fact. The patient was an old lady, seventy years of age, who resided at a considerable distance from the infected district. She had for some time previous been confined to her house, and had seen no one who had mixed with the sick, and consequently, had in no way been exposed to any of the real or reputed causes of the disease, of which, indeed, her attack presented few of the ordinary pathognomonic phenomena. She was seized very early in the morning with violent colic, attended with fever, great tenderness over the abdomen, flushed face, &c. She was bled at 10 o'clock; at 11, vomited a large quantity of coffee-ground matter, and died in about twelve or fifteen hours from the commencement of the attack. The next morning her body was examined by myself, in the presence of several of the most respectable and experienced physicians of the city, Drs. Otto, Hewson, Gallagher, and Monges, who all coincided in the opinion that the matter vomited, as also that which continued to be discharged from the nostrils, bore all the characteristic appearances of that ejected in the yellow fever—a disease with which they were all perfectly familiar.

“The stomach, as well as the intestines, was found to contain a large quantity of a similar substance. The cavity of the peritoneum being likewise filled with a large portion of it, we at first suspected the existence of an opening in the intestines, by which an effusion had taken place. After a careful and minute examination, however, no such opening was discovered. Our attention was now directed to the condition of the peritoneum itself, which was highly inflamed. It was, moreover, found that the substance in question exuded from its surface, the membrane, in many places, especially the portion of it which

¹ North American Journal, ii. 59.

² Deveze, p. 66; Lewis, N. O. Journ., i. 300, iv. 159; Rufz, p. 19; Kelly, Am. Journ., N. S., xiv. 383; Harrison, New Orleans Journ., ii. 138; Nott, Am. Journ., N. S., ix. 280.

³ Lewis, N. O. Journ., i. 300; Ib., iv. 159; Palloni, p. 10.

⁴ Bache, Am. Journ., July, 1854, p. 123.

⁵ Parrish, loc. cit., iii. 142; Merrill, N. O. Journ., viii. 6; Palloni, p. 10.

covers the liver, being coated so thickly with the grounds, that these could readily be scraped off with the back of a scalpel.”¹

With the preceding facts and considerations in view, there can be no difficulty in perceiving the impropriety of regarding the black vomiting as vitiated bile, and in acknowledging its being formed in the stomach itself. Nor is it less certain that an attentive examination of the subject will lead to a rejection of the opinion which ascribes this fluid to the secretory action of the diseased vessels of the stomach and intestines.

1. Before we can adopt this opinion with any show of reason, it would be necessary to explain satisfactorily how the same fluid, or fluids bearing so strong a resemblance to each other as to be regarded as identical by the most competent judges, can be secreted by the capillaries of parts differently organized, and which in their natural state, or during the course of ordinary diseases, produce fluids of dissimilar appearance or nature. If the black matter ejected from, or found in, the stomach and intestines is the result of a secretory process, the same kind of fluid issuing not only from other portions of the mucous surface, but from the skin and serous membranes, must be a secretion too. And yet these parts differ widely from the former, both as regards their organization and the nature of their ordinary products in health and disease.

2. Dr. Nott has well remarked, in objection to the theory before us, that the black vomit is most commonly seen in little particles or masses of various magnitude, which could not pass through a secretory capillary.²

3. It is admitted that the villous coat and the glands situated beneath it—the intervascular cells and their nuclei—are alone endowed with the secretory power in the stomach. Now, facts will be adduced, as we proceed, showing that the black vomit is unconnected in its formation with those parts; while, by the advocates of the theory of secretion, it is referred to, and, as we have seen, has been detected in capillary vessels which are not endowed with any power of the kind.

4. Such a detection affords no argument in favour of the theory of secretion; for, were the black vomit the result of such a process, it could not be found in the vessels of the circulatory system; for secretions, as is remarked by Dr. Michel, “are never seen in the bloodvessels of a part, not even with the aid of a magnifying power. The function is the result of the vital processes of the parts above mentioned, which elaborate the materials they derive from the blood into different products, then burst and discharge them.”³ It is evident, therefore, that if black vomit were the product of an operation of the kind, it would not be found collected in the capillary vessels, or that it would not be the means, by its discharge, of relieving the engorgement of these.

5. The very large, and even extraordinary quantity in which the black vomit is sometimes ejected, would alone constitute a strong objection to the theory of secretion; as we can find nothing to be compared to it in the healthy or diseased operation of any regular glandular or secretory structure.

¹ Monges, *op. cit.*, p. 60.

² *Loc. cit.*, p. 281.

³ *Charleston Journ.*, viii. 347.

6. The difficulties by which the theory of secretion, as connected with the formation of black vomit, is encompassed, are not removed by calling the fluid a sanguineous secretion, as the process by which such a secretion is produced, and the nature of the parts in which it is elaborated, cannot differ much from what obtains in regard to products having less or none of the characteristics of blood. Neither in the one case nor in the other can the fluid be formed by the capillaries. It may be remarked that, by the advocates of the secretory nature of the black vomit, no stronger reasons in favour of their views have been assigned than that the fluid differs somewhat from ordinary blood, and that it often presents a thick consistence and adheres to the inside of the stomach, forming a black coating of considerable thickness, and that it is found after death collected in the capillary vessels of the affected organ. But, besides that, it can be shown that the black matter is formed under circumstances precluding all idea of its being the result of a secretory process; the facts alluded to may be more readily explained by referring the formation of the fluid to the operation of other agencies.

7. The matter of the black vomit bears no resemblance to any known product of glandular action.

8. It is only met with in that state of the system, and that stage of the disease, when the secretory process in all other parts is generally or often suspended, or greatly impaired.¹

9. It not unfrequently happens, that the stomach or duodenum contains coffee-ground matter, while the intestines are more or less filled with coagulated blood.

10. In some instances, discharges of black vomit and of blood alternate; in others, again, the black vomit is found mixed with blood in the stomach after death; in another set of cases, coagula of blood are found in the intestines—"the surface having the appearance of the common black matter, while on cutting into them the centre is found to consist of red solid coagulum." "In such cases," as Dr. Monges remarks, "in order to adopt the opinion of secretion, we must believe that the same vessels occupied in the secretory process, afforded at the same time passage to a portion of common blood; for we can hardly admit that the mucous follicles are the organs secreting the black matter."²

11. Nor could such a product as the black vomit—supposing it to be a secretion—be thrown out from a surface as little altered in appearance as is the mucous membrane in a large number of instances. There is undoubtedly diseased action going on in the stomach—but, usually, this amounts to disturbance of function only, and not of structure—a condition of parts perfectly compatible with the formation of black vomit. "It does not," as is remarked by Dr. Michel, who has called attention to these facts—"it does not by any means imply organic lesion, without which destruction of the tissues themselves to a considerable extent, it could not exist as a secretion;

¹ Lewis, N. O. Journ., iv. 159.

² Op. cit., p. 61.

for I believe that a *secretion* of such appearance as the vomit, would plainly betoken a state of disease reaching even unto sphacelation.”¹

Enough has now been said to prove beyond doubt that the black vomit is neither altered bile nor the result of a secretory action on the part of the gastro-intestinal capillary vessels. In view of this, and taking into consideration various facts to which attention is next to be called, we shall be led to the conclusion that it is nothing but blood in a peculiar state of alteration. That it is the product of the stomach itself, when ejected from that organ, or of other surfaces whence it may proceed—the intestinal, urinal, pleural, peritoneal, &c., has been shown. That the black vomit, or a fluid like it, proceeds from the capillary vessels of these parts; that it may be, and has been pressed out of those vessels after death, and is often seen then and during life oozing out of the capillaries of parts accessible to sight, as the fauces, nostrils, skin, and eyes, are facts which, as we have seen, are too well established to be denied, and must of themselves go far to prove the sanguineous nature of the fluid, especially when we bear in mind the reasons assigned for rejecting the idea of its being the product of a secretory action of those vessels. For, if we refuse to admit that the fluid issuing from or pressed out of these has been secreted by them, we have no alternative but to regard it as blood; the only substance contained in those vessels, whether in health or disease. To the same conclusion we naturally arrive when we take into consideration the fact that the black vomit is the product, in some cases, of gastric inflammation occasioned by the ingestion of acrid poisons, or by other causes; and we know that in inflammation resulting from such agencies—or indeed from any agency—the vessels are engorged with blood, and nothing else; and that, therefore, what proceeds from them, whatever peculiar appearance it may assume, must be blood also. The same must be true as regards the contents of the capillary vessels in inflammation or congestion of the stomach or other parts in yellow fever. If we find these filled with red or dark fluid, and if their engorgement and dark colour are relieved by the discharge of that fluid, we may safely infer that the latter was blood, for hemorrhage is one of the common modes in which inflammation and congestion have a tendency to terminate; and if the red or dark fluid which occasions the relief in ordinary cases is admitted to be of the nature in question, there can be no reason to doubt that, when the effect is obtained in yellow fever, the fluid discharged is of similar kind. Nor is this all. We have seen that black vomit very generally adheres to those portions of the mucous membrane of the stomach or intestines that are inflamed or congested, or from which it was effused, leaving other parts uncovered. In this, we have a striking proof of its analogy to real blood, which exhibits a like disposition to adhere to inflamed surfaces from which it had exuded.

Again: We have seen that vomiting of black matter sometimes alternates with the discharge of pure blood, the ejection of black vomit preceding that of blood, or the reverse; that while the stomach or duodenum contains the

¹ Op. cit., viii. 347, 348.

former, the intestines are filled with coagulated or fluid blood; and that, in some cases, portions of fluid, more frequently of coagulated blood, are found, the surface only of which presents the granular appearance.

These facts cannot fail to point out the blood nature of the coffee-ground matter constituting the black vomit; inasmuch as it is not probable, as already remarked, that two distinct fluids—the one blood, and the other a secretion—should be thrown out from the same vessels in rapid succession, or simultaneously, or that the one should be smeared over by the other. Dr. Lewis remarks of the two fluids: “They are thrown up together, run into each other by perceptible degrees, just in proportion to the quantity of blood exhaled, or as it is intermixed with the secretions of the stomach and intestines.”¹ The same writer states the following fact. After remarking that in a large proportion of cases unchanged blood is vomited, he adds: “We have seen three brothers vomiting at the same time; one of them, slightly coagulated blood, resembling wine lees; another, fresh blood; and the third, the coffee-ground vomit. The young men were all purging blood at the same time.”² Is it probable, that while in these three cases, the intestines were giving vent simply to blood, the stomach in each should have furnished a distinct fluid? More natural is it to conclude, that this fluid in all was fundamentally the same. It may be stated, in addition, that blood which, when ejected from the stomach, presented its usual appearance, has been occasionally found to assume the peculiar characteristics of black vomit after being exposed some time to the influence of the atmosphere.³ Let me remark, besides, that black vomit imparts to muslin or paper a red colour, very similar to that resulting from dark blood.

That black vomit must be regarded as consisting in great measure of altered blood is still more satisfactorily proved by the fact that the latter may be, and has often been, converted artificially into a fluid bearing the most striking analogy to that ejected in the closing stage of the yellow fever. This fluid, as we have seen, contains usually, if not always, a free acid—the hydrochloric—the presence of which is evidenced by the taste as well as by the effervescence produced with the alkaline carbonates and other chemical tests. Now, if we impart to pure blood the same property by the addition of the aforesaid, or indeed of any other acid, we shall find it to assume, to a greater or less extent, the characteristic appearance of the black vomit. These results were pointed out long ago. Dr. Cathrall, the account of whose experiments has now been before the public more than half a century, while advocating a theory adverse to the one contended for, admits that, when blood is combined with such acids, “and the mixture diluted with an infusion of green tea, it resembles in many respects the black vomit;” and that “the odour arising from this combination so much resembles that arising from black vomit which has been kept for several years, that he could hardly distinguish one from the other.”⁴ The same experiment, slightly varied, has

¹ New Orleans Journ., iv. 159.

² *Ib.*, iv. 159; *Ib.*, i. 300.

³ Rochoux.

⁴ Essay on Black Vomit, p. 26. See Trans. of Philos. Society.

since been performed, and with similar results, by Dr. Lyon,¹ by Dr. Hope,² by Dr. Stevens,³ by Dr. Riddell,⁴ and others.

Dr. Nott, of Mobile, took a few drachms of blood from the heart of a patient dead of yellow fever, and added to it four or five drops of muriatic acid, diluted with a drachm or two of water, and shook them well together; the black colour was produced instantly.

“The same experiment was tried repeatedly on the blood of yellow fever patients, and on that drawn from a patient with pleurisy by cups, and the effect was invariably the same. Any one,” he adds, “wishing to form a correct idea of black vomit, has only to treat blood in this way, and add a little gum-water or flaxseed tea to represent the mucus of the stomach, and his curiosity will be gratified; and no one can tell the artificial from the genuine black vomit.”⁵

“I once, with Dr. Thomas Hunt, of this city,” says another distinguished American writer, Dr. Harrison, of New Orleans, “performed the following experiment: “A man was brought into the dead-house, while we were there. Upon examination, there was no black vomit in his stomach, but a whitish acid-smelling liquid, amounting to about half a pint. Into this stomach, containing this liquid, some blood from the vena cava was poured. At first, we thought the experiment had failed, and we returned to other investigations. Upon examining the fluid, however, after the lapse of ten or fifteen minutes, it was impossible to distinguish it from specimens of black vomit with which we contrasted it.”⁶

A late writer on the yellow fever, Dr. Blair, remarks that “blood allowed to drop, as in epistaxis, into water acidulated with muriatic acid, forms a very tolerable specimen of some descriptions of black vomit.”⁷ Dr. T. Y. Simons, of Charleston, while Professor of the Practice of Medicine in the medical school of that city, in 1833, was in the habit of performing the experiment of making artificial black vomit before the class in attendance.⁸ Dr. Michel, of the same city, remarks that, by carefully adjusted proportions of acid, it is possible to obtain all gradations of shades in the blood.

“But what is still more satisfactory in the experiment, the blood immediately curdles, as it were, into little flocculi, hardly dissimilar from those of the black vomit. It is difficult always to produce the coffee-ground sediment, since it is impossible to apportion certain particles of blood to those of the acid, as when it oozes in disease from the tissues. I have, however, been able to produce artificial black vomit so perfectly similar in this respect to that fluid itself, that their identity was at once apparent. During my attendance on a case of hæmoptysis, in which slight mouthfuls of blood were coughed up at a time, I obtained about a drachm, which was added to about

¹ Lond. Med. and Phys. Journ. for 1828, N. S., iv. 100.

² Cited by Dr. Lyon.

³ On the Blood, pp. 360, 422.

⁴ New Orleans Journal, ix. 420.

⁵ On the Pathology of Yellow Fever, Am. Journ., ix. N. S. 282.

⁶ New Orleans Journal, ii. 148.

⁷ Yellow Fever of British Guiana, p. 81.

⁸ Michel, Charleston Journal, viii. 343.

six ounces of water with a few drops of hydrochloric acid, which, upon being well shaken, developed all the characteristics of black vomit. It was allowed to rest, when the black flocculi formed the usual sediment."¹

The same effect is obtained by other acids, by chloride of platinum, weak tincture of iodine, and other substances.² Sir William Pym relates that, being attacked with hemorrhage from the fauces (in 1806), this was attempted to be stopped by applications of spt. terebinth. Having swallowed much blood and turpentine, he threw it up, and was struck with the resemblance it bore to black vomit.³

The change in question, and from the same cause, has been observed under other circumstances. Dr. Carswell, in experiments made several years ago, on the chemical dissolution or digestion of the walls of the stomach after death, found that, in cases in which the coats of the stomach were dissolved by the action of acids, a peculiar change in the colour of the blood contained in the veins of that organ had occurred. It was observed to accompany the chemical dissolution of the walls of that organ, and though less frequent in its occurrence than the latter, was not less obviously the effects of the same chemical cause, for there was no discoloration of the blood when there was no softening or dissolution of the coats of the stomach; 2, the discoloration of the blood was observed only in the vessels situate near the parts softened; and 3, both changes were, *cæteris paribus*, nearly in the same ratio as to degree and extent.⁴ The same able physician elsewhere calls attention to the fact that if we kill an animal—a rabbit, for example—or open it while alive, and during the process of digestion, the fundus of the stomach is always found to contain a quantity of gastric acid; and remarks that, “if arterial or venous blood is allowed to flow into the stomach from some of the neighbouring vessels, so soon as it comes in contact with the digested portion of the food, or that in which the gastric acid is most abundant, its natural colour is almost immediately changed to deep brown or black. The rapidity with which this change takes place in the colour of the blood, as well as the degree and extent to which it proceeds, varies with the strength of the gastric acid and the quantity of the blood on which it is made to act. When,” he continues, “the digested acid fluid, or the gastric acid alone is removed from the stomach in which it was found, and is put into another stomach, or other organ, the vessels of which are conspicuous and filled with blood, this fluid very soon undergoes the same change of colour which we have said takes place when it is poured upon the digested food, and consequently, when in immediate contact with the chemical agent by which this change is effected.”⁵

But this must suffice. Well may it be remarked that the production, in

¹ Michel, Charleston Journal, viii.

² Riddell, loc. cit., p. 420.

³ Bulam Fever, p. 223.

⁴ Rech. sur la Dissolution Chronique ou Digestion des Parois de l'Estomac après la mort. Journal Hebdomadaire, 1830, No. 87, p. 350; No. 91, p. 517; Cycl. of Pract. Med., iii. 99.

⁵ Cycl. of Pract. Med., article Melanosis, iii. 100.

this manner, of a fluid, the properties, colour, and appearance of which are in so remarkable a manner similar to black vomit, certainly constitutes a most ingenious experiment—one, indeed, so far as ocular demonstration of a general character is concerned, which is perfectly conclusive. (*Michel.*)

Evidently satisfactory, however, as the closeness of the analogy of the artificial black vomit to the true is, and recognized though it be by individuals—not one, but many—whose familiarity with the characteristics of the latter could not but be a warranty of the correctness of their opinion, it has been denied; and there are not wanting those, among intelligent observers, too, who maintain the possibility of pointing out a difference between the two fluids. In evidence of this dissimilarity, Dr. Kelly remarks that, while the true black vomit has a flaky appearance, “like smoky mica,” the artificial has more of the coffee-ground appearance; the addition of a certain quantity of sulphuric ether to the former will cause it to float on the surface, “having the appearance of a dark cobweb or flake of dead matter; while, by this means, the artificial is made to sink to the bottom, never more to rise. Nitrate of silver changes the black vomit “to a cream-white colour, every dark speck being removed, while the artificial substance retains its colour, being precipitated to the bottom of the vessel. Besides, if black vomit consisted of blood changed by the action of an acid, it is difficult to account for those cases in which, at the same time that that fluid is ejected from the stomach, pure blood is discharged from the bowels; inasmuch as we can see no reason why the acid secretions acted upon the blood in the stomach and not in the intestines.”

Plausible as all these objections may appear, they will be found, on close examination, to be but little calculated to shake our belief on the subject of the close analogy in question. If Dr. Kelly has not been able to obtain the flaky deposit in the artificial black vomit, and found that the latter has more the coffee-ground appearance, Dr. Michel arrived, as we have seen, at different results, and indeed found it “difficult always to produce the coffee-ground sediment.” Dr. Nott² also remarks that, by the addition of acid to blood, we do not obtain coffee-grounds, “which are due to the coagulation and agglomeration of particles.” Besides, even were it true, that these coffee-grounds are alone procured, the analogy would still hold; for the real black vomit often presents no other appearance; and all that could be said on the subject is, that by the means in question we can imitate one of the forms of black vomit, and not all. If, in some cases, black vomit is ejected from the stomach, and pure unaltered blood from the bowels, it is, as we shall see, simply because the effused fluid does not encounter in the small intestines, whence those discharges take place, the same modifying agencies it meets with in the stomach. Furthermore, whatever may be the difference as regards the effects of sulphuric ether and nitrate of silver on the real and artificial black vomit, the question of the identity of these is set at rest by the results of microscopical examination; for an account of which we are

¹ Am. Journ., N. S., xiv. 384, 385.

² Ibid., ix. 282.

principally indebted to Dr. Riddell,¹ of New Orleans, and Dr. Michel,² of Charleston. In speaking of the appearance exhibited by the artificial substance, the latter gentleman says in conclusion: "The blood was so dissolved as to colour the mass as usual, and the entire aspect was, in every particular, so completely analogous to black vomit, that an observer must inevitably be deceived as to whether he is examining the true or artificial product, for they are microscopically the same." Similar results have been obtained in this city.

But admitting that, notwithstanding all that has been adduced in favour of the opinion that the black vomit consists in altered blood, doubts on the subject may still be entertained, all difficulty must cease when the fluid is submitted to a chemical, and especially to a microscopical examination.

For the purpose of settling the first of these questions—the chemical composition of the fluid—and ascertaining how far its peculiar characters depended on the presence of blood, I placed in the hands of my friend, Professor Rogers, of the University of Pennsylvania, several specimens of it, for which I was indebted to the kindness of another friend, Dr. Cain, of Charleston, and subsequently other fresh portions obtained in this city during the prevalence of the fever in 1853 and 1854. In all the samples, except one, albumen was detected, and this was not examined for it, since it was impossible to free the liquid from the large amount of dissolved colouring matter which rendered it opaque. Sulphuric acid and chlorine were present in all. Iron was detected in the ashes of all. Alkaline bases were found in the ashes of all. Lime was detected in all, as also hydrochloric acid in the free state. The liquids were quite dilute, as their specific gravities, here given, show. Therefore, as the quantities furnished scarcely admitted of so minute an analysis, they were not examined for phosphoric acid.

Sp. gr. —, 1.016; —, 1.005; —, 1.004; —, 1.003.

The fluid obtained in this city gave the following results:—

Albumen.

Sulphuric acid in a state of combination.

Chlorine, " " "

Alkaline bases " " "

Earthy phosphates.

Iron.

Hydrochloric acid in the free state.

In his communication to me on the subject, Professor Rogers says: "These substances, although not the sole ingredients of blood, are yet all of them, with the exception of free hydrochloric acid, constituents of that liquid—a fact which, taken in connection with the other characters, and especially the microscopic appearance of the liquids, gives strong evidence that they contain much altered blood. Indeed, the presence of several of the substances enumerated, as albumen, iron, and sulphuric acid, seem not to admit of any other explanation, since it could scarcely be possible that in

¹ New Orleans Journal, ix. 420.

² Charleston Journ., viii. 344.

that stage of the disease they were the results of any food remaining in the stomach."

As regards the specific gravity of the specimens examined by Professor Rogers, it varied, as will be seen, from 1.003 to 1.016. In the examinations by Dr. Michel, of Charleston, it varied from 1.010 to 1.027—water being reckoned as 1.000. When kept until decomposition began, and bubbles of air and gases were produced, this changed very much, being sometimes as low as 0.986 or 0.962. The decanted liquid, without the solid parts, gave 1.000 to 1.015. A specimen examined by Professor Hume, and which was remarkably black, had a specific gravity as high as 1.032.¹

Of the composition of the white and precursory vomit, we know, as yet, little. Though some was collected on several occasions in the summer of 1853, the opportunity of having it chemically examined did not present itself. As already stated, it was strongly acid; and everything leads to the opinion expressed by Dr. John Davy, who remarks that, "judging from analogy, it will be found to be serous, or to contain albumen—the analogy, for instance, of the discharges in cholera, in diarrhoea, and dysentery, and, I may add, in catarrh. Even in common catarrh, I have found the perfectly transparent fluid which drops from the nostril, possessed of the properties of very dilute serum, exhibiting, with test papers, an alkaline reaction, and affording, on being boiled, a minute quantity of coagulated albumen."²

The black vomit has been submitted to a microscopic examination by Dr. J. Davy,³ Drs. Hayne⁴ and Michel,⁵ of Charleston, Prof. Riddell,⁶ of New Orleans, Prof. Alonzo Clark,⁷ of New York, Dr. Hassall,⁸ of London, Drs. Th. H. Bache, J. E. Rhoads, James Darrach, and J. Green,⁹ resident physicians of the Pennsylvania Hospital, and, at my request, by Prof. Leidy,¹⁰ of this city, whose skill and accuracy in matters of this kind are well known throughout the vast expanse of this country and of Europe, and the result of their experiments goes to prove, beyond doubt, the correctness of the views here advocated respecting the nature of the fluid, for they show the latter to contain some unchanged blood-corpuscles—a much larger mass of modified and disintegrated blood-corpuscles—epithelial scales—the scaly, columnar, and spheroidal, with their nuclei and nucleoli—granular detritus and irregular masses, apparently the results of degradation of blood-corpuscles—scales of opaque matter—crystalline bodies—amorphous matter, and sometimes oil-globules,

¹ Op. cit., Charleston Journ., viii. 334, 335.

² Blair on Yellow Fever (Appendix), pp. 158, 159.

³ Notes on Blair, p. 81.

⁴ Charleston Journ., viii. 345.

⁵ Ibid., v. 748, 749.

⁶ N. O. Journ., ix. 420; Ib., Microscopical Observ. pertaining to Yel. Fev. 1854.

⁷ New York Med. Times, ii. 230.

⁸ Lond. Lancet, April, 1853, Am. ed., p. 325.

⁹ Observations on the Pathology of Cases of Yellow Fever admitted into the Pennsylvania Hospital during the summer of 1853, by T. H. Bache, M. D., Am. Journ., July, 1854, pp. 125–6. See, for other examinations, N. O. Med. News, p. 303 (1854).

¹⁰ See an Essay on the Black Vomit, by the author, Am. Journal, April, 1854, p. 312.

and torulæ, and spores. In a word, the microscope enables us to perceive that the sedimentary portion of the black vomit is composed almost entirely of blood-corpuscles in various stages of degradation.

The black liquid matter formed in the upper part of the intestinal canal, as well as the coffee-ground fluid discovered in other portions of the latter, has much the same appearance under the microscope as that which proceeds from the stomach. Somewhat different is the result obtained in the examination of the dark or black pasty and tar-like matter discharged from the bowels, and found, and doubtless formed, in the lower portion of the canal. Of this, the microscopic character bears but a slight resemblance to that of the gastric effusion. It is found to consist of a muco-granular substance, containing a multiplicity of lacerated cells, but no blood-corpuscles. Dr. Michel states that the dark-bluish mucous colour of this substance is hardly obscured by any hæmatin. True, however, as this may be, we can find no reason to doubt the identity or close connection of this fluid with the true black vomit, or to withhold our belief from the fact that the difference arises from the circumstance that, in cases in which the matter ejected per anum assumes the appearance in question, the blood has undergone a kind of digestion in its passage through the intestines.¹

Dr. Samuel Jackson, in his account of the epidemic of yellow fever which prevailed in this city in 1820, remarks that Dr. Rhees, the resident physician at the City or Fever Hospital, in some experiments he instituted on the black vomit with a solar microscope, found innumerable quantities of animalculæ to exist in it. A single drop contained many thousands, being apparently a congeries of them. The black mucus of the intestines exhibited the same phenomenon. When the matter fresh thrown from the stomach was examined, the animalculæ were alive and in constant motion; but if taken from the dead subject, or inspected after standing some time, they were always dead and quiescent.²

Others have made analogous statements, and very recently Dr. Manly, in a short communication to the *London Lancet*, remarks that his attention was first called to the subject by a paragraph in *Wood's Practice*, and that, during the prevalence of the yellow fever in Pernambuco in 1852, he verified the fact in several instances—the animalculæ being acari (species unknown).³

Nothing of the kind, however, has been found in the experiments of Dr. Clark, Dr. Leidy, and Dr. Hassall. Dr. Michel, in a short essay on the black vomit published in the fifth volume of the *Charleston Medical Journal*, stated that the fluid never presented infusoria when fresh, but that these were developed whenever small quantities were allowed to remain in the room (p. 748). Subsequent examinations, however, made on a larger scale, have not confirmed this statement; for Dr. M. now informs us that, in recent samples of the ejecta, he never detected spontaneous evolutions of the kind.

¹ Michel, loc. cit., p. 338.

² An Account of the Yellow or Malignant Fever, as it occurred in the City of Philadelphia in 1820, p. 81; *Ib.*, in *Phila. Journ. of Med. and Phys. Sciences*, ii. 23.

³ *London Lancet*, Am. ed., Feb. 1853, p. 192.

“On this subject,” he says, “I have experimented with care; and if by *animalculæ* we are to understand the initiatory forms of *animal* cell-life, as they are recognized in the familiar varieties of *polygastrica* and *rotifera*, I say, without hesitancy, that such were never to be seen, either in fresh or putrescent specimens of the vomit. Dr. Rhees’s experiments were made with the solar microscope, an instrument certainly not adapted for such investigations as these.” “If infusoria were present, we could hardly fail to encounter them; for whoever has watched the development of the simple monad, is aware that they are propagated with such magical rapidity, that when one is discovered, countless numbers are soon found to follow in its suite.”

In the experiments of Dr. Riddell, minute filiform algæ were uniformly present, varying from .00001 to .00003 in thickness. Several jointed forms of moving algæ were equally constant, though mostly very small. Vital organizations still more minute were constantly met with. In one sample of black vomit, an unusually trifling form of that rather rare parasitic organism, the *Sarcina*, was observed. The cells or segments in that sample were arranged mostly in squares, and each separate segment, having an oval form, measured near .00003 (p. 421). Other investigators have found nothing of the kind. Nor is this to be wondered at; for, as has been remarked by both Dr. Hassall and Dr. Michel, the black vomit, from its intense acidity, is not favourable to the development of algæ, which are not likely ever to be present except as the result of decomposition. From the same cause—the acidity of the fluid—and from the circumstance of the latter being an organic product, we must be better prepared to find it containing fungi of various kinds. Hence, these products—especially the *Torula*—have often been described as present. A small variety of the concentrated form of them was discovered, with one or two exceptions, in every sample examined by Dr. Riddell. They were generally very abundant. The individual buds were oval, averaging .00018 in length by .00012 inch in breadth. The examinations of the putrescent fluid afforded Dr. Michel an opportunity of witnessing the development and mode of growth of a fungus protophyte very much resembling the *Torula cerevisii*, if it be not identical; the only difference being the constant absence of nuclear cells at all periods of its development and growth. Dr. Hassall detected—1. Ramose branches of the sporules of a fungus. 2. Large circular sporules, usually single, but sometimes ranged in rows, and giving origin to slender threads or filaments. 3. Branched and moniliform threads of a fungus usually occurring in bunches. 4. Many compound cells, having the appearance of sporangia. Dr. Clark, on the other hand, found in one of the specimens he examined a vegetable growth, in linear or rather stint joints, the sections commonly separated, but often united as by a hinge. It was in great abundance, but disappeared entirely after the lapse of three weeks. The same was found by Dr. Riddell.

It thus appears that different fungi are evolved under particular circumstances, for each of the investigators referred to has seen different growths; while in many instances they fail entirely to be observed. From this we conclude that, when these bodies are found, they are accidental, and may not

improbably be referred to particular substances administered as food or medicine, and that consequently, contrary to the opinion of Dr. Hassall, they have no relation to the cause of the vomiting.

But be this as it may, the inquirer who bears in mind what has been said respecting the microscopical characters of the true black vomit, backed by all the corroborative facts and circumstances before adduced, and who, at the same time, takes into consideration the close analogy existing between the fluid ejected from the stomach and other parts and that artificially made by the means mentioned, will, unless labouring under the incubus of some preconceived and favourite theory, feel little disposition to deny or doubt the sanguineous nature of the substance in question. It is altered blood mixed with inspissated viscid mucus and other gastro-intestinal secretions, themselves modified by a larger or smaller portion of hydrochloric acid.

The next question which presents itself is, how and where does the blood thus effused undergo those changes which impart to it the characteristics of the black vomit?

It has been supposed that the blood thus discharged in the cavity of the stomach has coagulated there, or on the surface over which it was effused, "and, having been detached and triturated by the violent and frequent contractions of that organ, in the efforts to vomit, has had its appearance as a coagulum of blood altered, and its colour darkened by the gastric juice, or by some chemical decomposition, either spontaneous or produced by the action of the air, or other matters contained in the stomach."¹ Others have attributed the effect to the blood's being brought under the digestive power of the stomach.² That this opinion receives support from the facts mentioned of blood ejected from the stomach assuming subsequently the character of black vomit, and of coagula being found in that cavity presenting only exteriorly these characters, may perhaps be true. But it is opposed by facts and circumstances entitled to consideration. The stomach, at the period when black vomit is ejected, is not usually in a condition to carry on the digestive process; while in hæmatemesis, where the organ is comparatively healthy, or at least has not undergone changes calculated greatly to impair its digestive functions, the blood, though dark in colour, rarely assumes the coffee-ground appearance and other characteristics of true black vomit. Furthermore, in yellow fever the matter seldom remains long enough in the stomach or bowels, or on the surface of the membranes, from the capillaries of which it is effused, to be changed by the digestive process, supposing this to be less impaired than we know it to be, and to undergo much of the trituration referred to by Dr. Bancroft. We can sometimes, as Dr. Evans remarks, distinguish this organ becoming more and more distended under our fingers as the matter accumulates, which it does in about ten minutes, and sometimes in a shorter period; and when filled the vomiting is renewed.³ In addition

¹ Bancroft on Yellow Fever, p. 28.

² Audouard, *Recueil*, *Introd.*, p. 1; Rochoux, *op. cit.*, p. 533.

³ A Clinical Treatise, &c., p. 249.

to this, it may be urged that the matter of the black vomit oozes at times from surfaces where it can have undergone, or can undergo, no digestive influence, or no trituration; and has, as stated above, been detected in the engorged capillaries of the stomach and intestines, where, of course, it could by no possible means have been formed by the process contended for.

More natural is it to conclude,¹ with many modern writers, that the blood acquires generally, if not always, in the stomach and intestines—or in whichever part the phenomenon is observed—its peculiar and characteristic features; and that, when it does so, the effect, as may be inferred from the almost universally acid nature of the vomit, and the well-ascertained conversion of blood into a substance perfectly identical with that fluid by the addition of acids, is doubtless due to its meeting there with a large portion of free hydrochloric acid, the morbid product of the disease.

Dr. Carswell claims the credit of being the first to suggest or prove the correctness of this opinion. “The fact of the black discoloration of the blood effused into the cavity of the stomach and intestines being produced by the chemical action of an acid fluid or gas contained in those situations, does not appear to us to have been ever suspected till after the results of our experiments on this subject were made known.” “The matter of black vomit and dejections was *believed* to be *altered* in the stomach and intestines; but whether by a chemical or vital agent was quite undetermined.”²

In relation to this matter, it may once more be remarked that Dr. Cathrall, more than half a century ago, was fully aware that, by the action of an acid, blood was changed in its characters, and converted into a substance resembling in every particular the genuine black vomit. Before him, Portal, in speaking of the appearance of blood effused into the cavity of the stomach, remarked that doubtless it there “assumes a dark colour; because, not being then in contact with oxygen gas, it becomes carbonized by combining with the carbonic acid which exists in the stomach and intestines.”³

Dr. Samuel Brown, of Boston, in his account of the epidemic which prevailed in that city in 1798, in speaking of the septic acid, which, according to Dr. S. L. Mitchell’s theory, constitutes the cause of the disease, and is formed in the *primæ viæ*, as well as derived from external sources of organic decomposition, says: “The coffee-coloured matter, commonly called black vomit, ejected in what are called bilious remitting fevers, seems to owe its colour to a mixture of this *acid*, as appears from its stimulant nature noticed by dissectors, with a quantity of bile and *blood*, which is poured out of such vessels as have their coats destroyed by this poison.”⁴ Dr. Lyons, as already stated, published in 1828 the opinion, based on experiment, that black vomit

¹ Nott, p. 281; Am. Journ., ix. 282; Blair, p. 81; Wood, i. 301; Lyon, loc. cit.; Riddell, loc. cit.; Hunt, quoted by Dr. Riddell; Stevens, 288, 330; Michel, op. cit. 343, 344; Carswell, Cyclop., iii. 102; Pym, op. cit., p. 222; Harrison, N. O. Journ., ii. 147.

² Cyclop., iii. 102.

³ Cours d’Anatomie Médicale, v. 188; Ib. Obs. sur la Nature et le Traitement du Méléna—Mém. sur plusieurs Maladies, ii. 209.

⁴ A Treatise on the Nature, Origin, and Progress of the Yel. Fever, &c., p. 46, 1800.

was blood changed from its normal state by the action of muriatic or gastric acid ; and Pym, whose volume on the Bulam fever appeared in 1814, states positively that black vomit is blood modified by an acid. The account of Dr. Carswell's experiments appeared in 1830.

The propriety of this view of the mode of formation of the black vomit is farther illustrated by phenomena observed in other diseases, in not a few of which the changes in the blood, which approximate it in a greater or less degree to the fluid in question, are evidently due to the action of the acid secretions with which it comes in contact, or is mixed. The dark colour of the blood in hæmatemesis, which at times contrasts so strikingly with the florid blood of hæmoptysis, has usually been ascribed to the circumstance that while the latter is of arterial origin, the former proceeds from the venous capillaries. With much show of reason it has been suggested that the difference depends on the circumstance of the admixture of the blood on the one hand, with oxygen in the lungs ; on the other, with the hydrochloric acid of the gastric secretions. The probability of the fluid and gaseous contents of the alimentary canal acting on the blood after it is poured out, and depriving it of its bright colour, was strongly insisted upon by Portal (see the celebrated work just quoted), who disbelieved, as Areteus had done long before him, the venous origin of hæmatemesis ; an opinion in which that very distinguished writer has been followed by several pathologists, and among these by our countrymen Drs. Condie, Nott, and Michel. If the comparatively small proportion of the acid existing in that disease can darken the blood to the extent often noticed, we can readily conceive how a much larger portion of it, acting on blood already impure, can impart to it the characters of black vomit. Attention has been called, in an early part of the preceding chapter, to the fact that a fluid in every respect resembling the black vomit, is sometimes ejected in dyspepsia and during pregnancy ; and every one knows that, in such conditions of the system, the predominance of acid in the stomach is a very usual phenomenon ; and on inquiry it will be found that, in those cases in which the effusion of this black substance took place, the acid formation was particularly noticeable. Dr. Michel reminds us that this again is seen in certain instances of renal affections ; the blood becoming ultimately mixed with the acid mucus of the bladder. The same writer, with great correctness, remarks that blood is frequently seen to be red while it oozes from the surface of the uterus, but becomes dark so soon as it combines with the vaginal secretions, which are decidedly acid.

In some of the varieties of what Dr. Carswell has denominated the spurious melanosis, the discoloration of the blood is satisfactorily shown to be the result of the action of chemical agents, the change taking place, as we have seen, in the vessels or cavity of the stomach, and in the cavity or vessels of the peritoneum.¹ Here, the modifying influence of gastric or hydrochloric acid is too apparent to be denied.

Doubtless we may explain in the same way the occurrence of the discharge

¹ Cyclop., iii. 99.

of blood similarly altered from other surfaces. The acid character of the sensible and insensible perspiration, under particular circumstances, is well known; and that the skin secretes at times hydrochloric acid, was several years ago ascertained by Dr. William Lyon, of Dominica, whose observations were made on recruits arriving in the colony.¹ With this fact in view, we can readily account for the black and grumous appearance which the blood effused from the denuded cuticle, in some cases of yellow fever, has been found to assume. May not something of the kind take place in the serous membrane, also, from the formation of acid in that tissue, or through the effect of endosmosis during life, or of imbibition after death? On this subject, we are not left to conjecture. Dr. Carswell, so often referred to, who has adopted and strongly advocated this opinion, and whose experiments on the discoloration of the blood and the dissolution of the walls of the stomach by the action of gastric acid are well known, found that the influence of that acid in producing the first of these effects, is not limited to the contents of the alimentary tube. Not only, he says, is the blood that has been poured into the cavity of the stomach, or is contained in the vessels of the mucous or submucous tissues changed from red to brown or black, but likewise that of the sub-peritoneal vessels of the same organ is similarly altered. Dr. C. has also seen the same black discoloration of the blood in the sub-peritoneal vessels of a neighbouring organ, such as the liver, spleen, intestines, or diaphragm in contact with that portion of the stomach which contained half-digested food or gastric acid. It is not difficult to understand that blood effused into the cavity of the peritoneum will undergo the same change of colour as that which takes place in the fluid contained in the vessels. Dr. C. produced effusion of blood into the cavity of the peritoneum around the stomach in several animals, and was thus enabled to witness its conversion from red to brown or the deepest black. He took a portion of stomach containing gastric juice, placed it on a coagulum of blood, or kept it in close contact with a portion of intestines on which there was a greater or less number of vessels filled with red blood. In both cases the blood assumed a brown or black colour, no doubt from the acid having been carried from the interior of the stomach by imbibition.²

But be this as it may, the formation by the gastro-enteritic mucous surface, in the progress of yellow fever, of an acid fluid of sufficient strength, and in sufficient quantity to affect the blood in the way which the same kind of agents do in the hands of the chemist, cannot be matter of astonishment. We know that such a fluid is always found in the stomach during the digestive process in the state of health. Dr. Prout, so early as 1823, announced as the result of his experiments, that such was the fact; and he found that this acid was free, or at least unsaturated, muriatic, or hydrochloric acid. Bernard, Thompson, and others, have thought that this acid was the phosphoric; by others it has been regarded as the lactic. But it may be safely said, that subsequent experience has confirmed the opinion expressed by Dr. Prout, as to the ex-

¹ Observations on Black Vomit, Lond. Med. Phys. Journ. (N. S.) iv. 100.

² Op. cit., iii. 100.

istence of the hydrochloric acid, the proceeds of the chloride of sodium of the blood, which, while it contributes that acid, at the mucous membrane of the stomach, to the gastric fluid, leaves free soda to be carried to the liver by the veins of the stomach. In fevers, acid is preternaturally formed. Dr. Stevens (p. 322), in speaking of a case of malignant lake fever, which he saw in 1830 at Rochester, N. Y., in company with Dr. Henry, a distinguished physician of that place, says:—

“The stomach was so excessively tender that the patient could scarcely allow even the slightest pressure to be made on the epigastrium. The tongue was exceedingly foul, and when I applied a small piece of moist litmus paper to this organ, the test was reddened almost as suddenly as if it had been dipped into a strong acid. The same thing occurred when litmus paper was dipped in the fluid ejected from the stomach.”

A similar change takes place, even more uniformly, in the yellow fever. It does so long before the appearance of black vomit; and we have seen that the white ropy fluid frequently ejected at the close of the second stage is usually intensely acid. Can we, when bearing these facts in mind, and recollecting the effect of acids on the blood *out* of the body, fail to discover a ready explanation of the formation of black vomit?

But it is not less possible that the change may take place, not in all cases, as some writers have affirmed,¹ but in some before the blood has been effused into the cavity of the organs, and consequently in the capillary vessels. Its discovery in these, as mentioned in a former page; its frequent and quick renewal; its effusion in an apparently formed state from external and visible surfaces, undoubtedly lend some support to the belief. But if such is the case, if the vessels through which the black vomit exudes, really modify the blood in its transit to the surface, and, in that sense, may be said to secrete the former—for the idea of a regurgitation, once entertained by or attributed to some of our physicians,² can scarcely be seriously thought of—they cannot, from all we have seen, be admitted to do so otherwise than through the agency of an acid, which must reach the blood by the process of endosmosis. Whether this suggestion will be ever confirmed; whether the acid, being in the stomach and intestines, finds its way into the bloodvessels which subsequently throw off the black vomit in its formed state; or again: whether the acid is formed in the very capillaries which contain the blood modified in the way some have suggested, remains to be verified; for, so far, these opinions are mainly conjectural. In the meanwhile we cannot go astray, if, with able pathologists and accurate experimentalists, we regard this change, when it takes place in the capillaries, as the effect of a cadaveric alteration. We have seen that, in the experiments of Dr. Carswell, a peculiar change in the colour of the blood contained in the gastric vessels is observed to accompany the dissolution of the coats of the stomach, and that this is not less obviously the effect of the

¹ Waring, *Fever of Savannah* in 1820, p. 52. Kelly, *op. cit.*, p. 385.

² Stevens on the Blood, p. 330.

same cause—the agency of the gastric acid. We have seen that, in some experiments, when the digested acid food, or the gastric acid alone, is removed from the stomach in which it was formed, and put into another stomach, or other organs, the vessels of which are conspicuous and filled with blood, the fluid very soon undergoes the same change of colour noticed when the chemical agent is placed in contact with it; and also that the same effect is produced in the peritoneum when a portion of the stomach, containing gastric juice, is kept in close contact with a portion of the intestine in which there was a greater or less number of vessels filled with red blood. In all these cases, the dark colour of the blood could not have been produced otherwise than by the passage to it of an acid by means of cadaveric imbibition. In corroboration of this, it may again be stated that, in the experiments of Dr. Carswell, the discoloration of the blood did not occur when there was no softening or chemical dissolution of the coats of the stomach; that the discoloration of the blood was observed only in the vessels distributed over or situated near the parts of the stomach which were softened; and that both changes were, *cæteris paribus*, nearly in the same ratio as to degree and extent.¹ It need scarcely be remarked that this kind of softening is cadaveric. To this, let it be added that, in the autopsies made by Dr. Michel in 1849, the bodies were opened immediately after death, while yet warm, and that this able physiologist never noticed the slightest trace of black matter in the vessels. The change, therefore, when it takes place, must do so after the acid mixed with the blood in the stomach has remained some time in contact with the lining membrane of the organ, and is the more readily produced because the absence by exfoliation of the epithelial covering, and the softening of the membrane, which so generally occur, increase the facility of the passage of the acid through the coats of the delicate capillaries.

The peculiar characters, and particularly the dark hue of the black vomit, may, and probably does, as well as its manifest disposition to exude from the capillary vessels, depend, in some measure, on the diseased condition of the blood prior to its mixture with the acid fluids of the stomach, or to its undergoing, while in the circulation, the modifying action of such acids. That this is the case, has been surmised by competent authorities,² and may be inferred from the fact that, at the period when the black vomit usually sets in, the blood is, in most cases, of a dark colour, and has lost a large share of its adhesive and coagulable properties; and that clinical experience and physiological experiments have shown that, when in that diseased condition, either from the agency of ordinary and powerful morbid and acrid poisons, or from the artificial introduction into the circulation of putrid substances, the blood manifests an equal disposition to exude from the same parts, and exhibits often many of the peculiar characteristics of the black vomit. Well founded, however, as this explanation may appear, it cannot be

¹ Op. cit., p. 100.

² Imray, p. 91; Evans, p. 249; Blair, p. 81; Levacher, pp. 82, 83; Pringle, p. 197; Hunter, p. 64.

concealed that it is not applicable to all cases; for we not unusually find genuine black vomit occurring contemporaneously with effusion of florid and coagulable blood from the nostrils or other parts;¹ a circumstance it would be impossible to account for, were an alteration of the circulating fluid essentially requisite to enable the latter to furnish materials towards the formation of the black vomit, and to impart to that fluid the disposition to escape from the capillaries. But such cases are comparatively rare. Most generally, black vomit is associated with altered blood, and the occurrence of the aforementioned cases will be found to be due to the early formation of acid fluid in the stomach or bowels, and the consequent transformation in those organs or their capillaries of the blood therein contained into genuine black vomit, while the rest of the circulation remains unimpaired, or nearly so.

It has been supposed that the discharge of the black vomit from the stomach, like that of the same matter, and of ordinary blood from other outlets in the yellow fever, is always the result of a morbid process of an active kind; in other words, that it is an active, not a passive hemorrhage.² And there is little doubt that this view of the subject receives support from the fact, that the effusion of the fluid is often preceded by an inflammatory irritation of the surface whence it proceeds, or by a greater or less inflammatory excitement of the system at large, both of which appear incompatible with the existence of an atonic state of the capillaries. Nor is it less certain, that a fluid exactly resembling the black vomit is, as we have seen, formed in cases of poisoning or external injuries, under the direct influence of active inflammation; and that, in some of these cases, recovery occurs, convalescence is rapid, and the patient is restored to health without having had recourse to tonics and stimulants, or any means calculated to correct the atony of the vessels.³ But the occurrence of such cases only proves that the effusion of a substance like the black vomit may take place under the influence of an active state of the capillary vessels, but not that it never occurs under the influence of a contrary condition of the same vessels. With these facts before us, we cannot admit that when, in the yellow fever, the black vomit occurs, the hemorrhage must be viewed as of an active kind. So far from this, the circumstances under which the discharge takes place in that disease; its connection with other symptoms, which portray an atonic state of the system; its analogy to other hemorrhages, undoubtedly of a passive kind, must lead to an opposite conclusion. The effusion of black vomit, as well as the discharge of blood from other surfaces, takes place generally at a period of the disease when the powers of the system are exhausted and all its vital energies are in abeyance, and when all irritation or inflammation, if it existed before, has subsided. In many cases, the existence of such inflammation prior to the effusion is problematical. The discharge is attended with passive exudation of a similar fluid, or altered blood, under the cuticle; it is accompanied with symptoms indicative of prostration and atony of the sanguiferous and

¹ Blair, p. 81.

² Catal, p. 11.

³ See, for such, Dr. Monges' Remarks, N. A. Med. and Surg. Journ., ii. 60.

other organic systems ; it is preceded and attended by other hemorrhages, the sources of which are visible to the eye, and in regard to the passive nature of which there can be no mistake ; while, in some instances, the exhalation and discharge of the fluid continue after death, when all conditions of activity have, of course, subsided ; and the limited number of cases that recover after the accession of the black vomit, do so generally under the influence of means calculated to restore tone to the capillaries, and to impart strength to the system at large.¹ From all this we may perceive the propriety of coinciding with those who regard the effusion as connected with a relaxed condition of the vessels on the surface whence it proceeds.

The black vomit being recognized to be blood acted upon by the acid contents of the stomach, we have no difficulty in perceiving that much of the difference it presents in regard to its physical appearance will depend on the manner in which the blood is effused into the stomach—whether drop by drop, or in a stream—and on the degree of acidity of the gastric secretion, or the quantity of serous fluid it meets in that organ. It has been found, for example, that whenever blood is exhaled therein in a quantity proportionate to these secretions, it exhibits a black colour, while the aqueous portion is limpid, or clear green. If there be a slight excess of blood—more than enough to neutralize the acid—instead of black, we have a nut-brown, a chocolate or reddish matter, and the watery portion, when filtered, is of a rum, brandy, or red colour. If the hemorrhage be great, the fluid presents all the characteristic marks of blood, either with or without admixture of black vomit. Dr. Nott² has often seen a tablespoonful or two of the “coffee-grounds” at the bottom of the basin with a pint or more of pure blood. The addition of a little bile, which in some cases doubtless occurs, will impart a greenish tinge to the aqueous portion. All these varieties of colour may be, and have been imitated in the artificial black vomit. A very small quantity of blood oozing gradually in a minutely divided form, and mingling slowly with the secretions of the mucous membrane of the stomach, will make a large quantity of black vomit. Dr. Nott says that, judging from his experiments, he thinks a tablespoonful would make a pint (p. 283).

While such is the appearance and the nature of the black matter ejected from, or found in, the stomach, the intestines give passage, as we have seen, to substances which bear a strong resemblance to the former, and are, to all intents and purposes, analogous to or identical with it. So far as the source of the black vomit is concerned, no difference of opinion at present exists. It proceeds from the lining membrane of the stomach itself, and from no other part. But in reference to the surface whence that passed per anum, or found in the intestines after death, proceeds, doubts have been entertained—some maintaining that it is the product of the intestines themselves, as well as sometimes of the glandular organs which are dependent on the latter ;

¹ Blane, p. 410 ; Chervin, Catel., p. 11 ; Imray, p. 91 ; Harrison, p. 148 ; Kelly, p. 385 ; Levacher, p. 82.

² Am. Journ., ix. 282.

others, that in general it has reached them through the pylorus from the stomach.¹ To this last source it has been more particularly referred when the discharge occurs towards the fourth day of the disease, or later; when it is voided some time after black vomiting has set in, and especially when, from the absence of symptoms indicative of inflammatory irritation or congestion, we may infer that the intestinal tube has remained unimpaired. In this opinion, however, no one who takes into consideration all the circumstances connected with, or attending the ejection of black matter from the bowels can concur. That the black matter found in or voided from the intestines is sometimes—we may even say often—of gastric origin, there can be no reason to doubt; for whenever the matter is limited to a part of the intestines, it is found only in the jejunum. But however true this may be; however reasonable or proper the admission may be regarded; and whatever may be the degree of reliance placed on the accuracy of the signs by which that origin may be inferred, we must not explain in that way all the cases of alvine discharges of black coffee-ground, or dark sanguineous matter which occur in that disease; for there are facts enough to show, beyond the possibility of denial, that such dejections are referable, in the greater number of cases, to morbid processes located in the intestines themselves and their dependent organs.² Admitting, as we are bound to do, that such evacuations consist in part of altered blood and vitiated secretions, usually mucous, with sometimes an admixture of bile; considering that some of these secretions proceed necessarily from the abdominal glands; that others are as naturally found in the intestines as in the stomach; and that there can be no reason to doubt the existence in the intestines, any more than in the stomach, of the hemorrhagic tendency so characteristic of the yellow fever, we might *à priori* conclude, that the ejections in question proceed from the former as often, at least, as from the latter organ; at any rate, that no reason exists why they should not do so. But we have stronger proofs than that. If the contents of the small intestines have an alkaline reaction; if the pancreatic juice which is poured into the duodenum is invariably of the same character; if, in these organs, no acid like the gastric is found to exist; and if, therefore, from the existence of the former chemical agents, and the absence of the latter, it may be argued that the blood cannot be modified in the way necessary to cause it to assume the character of black vomit, it may be remarked that, in lieu of gastric or hydrochloric acid, and to counterbalance the alkaline influences above noted, it encounters lactic acid in the cæcum, and sulphuretted hydrogen or carbonic acid in various portions of the tubes, all of which impart to it an acid reaction, and are thereby fully able to produce the changes in question.³

Black matter, having all the ordinary characters of black vomit, has been often found in the small and large intestines in subjects whose stomachs con-

¹ Rochoux, p. 360; Louis, p. 102; Michel, *op. cit.*, p. 346.

² Evans, p. 248; Arnold, p. 38; Nott, *loc. cit.*, p. 283; Louis, p. 103; Audouard.

³ Carswell on Melanosis in *Cyclop. of Pract. Med.*, iii. 101; Michel, *Ch. J.*, viii. 346.

tained none. Louis, among others, mentions two cases of the kind in which the small intestines were the seat of such an exhalation while the stomach was empty. One of these subjects never had vomited. He also mentions a case in which black matter existed in the large intestines, while the stomach was, as in the former, free from it.¹ Similar instances have been described or alluded to by Dr. Lewis, of Mobile, and others. In the case just referred to, says Lewis, "there was no black vomit, so that the black matter of the large intestines may be considered in this case as the result of the exhalation of the mucous membrane of the colon; and from the facts in this and the preceding articles, it would seem to follow that the mucous membrane of the stomach, that of the small and that of the large intestines, in an undetermined proportion, had given rise to the brown or black matter with which they were in contact."² It should be borne in mind, also, that clots of coagulated blood, smeared over with coffee-ground matter, are either passed from, or discovered in the intestines. The black matter sometimes comes in form of figured stools composed entirely of it, and made into form and consistence merely by a small quantity of intestinal mucus; the whole mass being, as I have already had occasion to mention, except in colour, of the usual appearance of a child's alvine evacuation.³ Nothing of the kind is found in the stomach, and as it is not likely that the effect noted could be produced by means of coffee-ground matter formed at a distance from the part where such compounds were found, or from which they had been discharged, we must view them as formed or covered with a matter effused by the intestines themselves. Nor must we forget that, if black vomit often precedes the ejection of black stools, the contrary is not unfrequently the case; the matter being evacuated from the bowels before it is ejected from the stomach, or indeed before it is *formed* in that organ, as proved by a comparison of substances voided both ways. All this proves, as Louis judiciously remarks, that the black matter does not come from one source only, but may come as well from the intestines as from the stomach. Moreover, the tar-like or coffee-ground matter has been found, after death, oozing from the vessels on the surface of the villous coat of the intestines and other abdominal organs; or it has been pressed out from congested portions of the intestinal membrane.⁴ Again, when black matter is voided per anum, the patient stands a better chance of recovery than when it is discharged by the stomach; a result which would scarcely obtain were it formed in the latter organ. For, were this the real place of its origin, the indications of danger would be as great when the matter is voided from the bowels, as we know it to be when it is vomited; there being no reason why danger should be lessened by the mere fact of the matter passing into the intestines instead of being expelled by the mouth. In conclusion, it may be remarked that the appearance of black coffee-ground stools towards the fourth day cannot prove the gastric source of the effusion, as it

¹ Researches on the Yellow Fever of Gibraltar, pp. 103, 112.

² Op. cit., p. 112.

³ Dickson, Am. Journ., ii. 73.

⁴ Arnold (of Jamaica), p. 38.

is about this period that the hemorrhagic tendency is established ; and lastly, that nothing can be made out of the absence of signs of intestinal inflammation or congestion, inasmuch as the effusion is the result of a passive or relaxed condition of the vessels, and, like black vomit, is perfectly compatible with a state of integrity of the mucous membrane.

CHAPTER XIV.

TONGUE, THIRST, RESPIRATION, AND PAIN.

a. STATE OF THE TONGUE.—In respect to the condition of the tongue, the yellow fever of this city approximates closely to that of other places both in temperate and tropical regions. Here, as elsewhere, that organ generally assumes at each stage particular appearances which serve, to a certain extent, to distinguish the disease from others of a kindred nature ; and here, also, these appearances differ somewhat in different cases, and at different epidemical periods ; but, at the same time, experience shows that here, as elsewhere, the condition of the organ, in the fever before us, proves, but too frequently, a fallacious guide in a diagnostical or prognostical point of view.

In very many cases, the tongue for some hours, perhaps a whole day, after the commencement of the attack, preserves its natural appearance.¹ At this period, and during the entire first stage, it is sometimes dry and hot, commonly, though not always, moist, differing in that respect from what usually occurs in febrile diseases generally ; for, in these, a dry tongue is a usual attendant on the stage of arterial reaction, or on local inflammation existing at that period. This moist tongue, which accords but little with the excitement of the pulse and the elevated temperature of the skin, with which it is often associated, was noticed by Dr. Rush, and others, in the epidemics of 1793 and 1794 ; and has since been found to exist in all our subsequent visitations,² and is pointed out by most writers as a very common symptom at this period of the disease.³

¹ Hillary, p. 150 ; Heustis, p. 110 ; Deveze, p. 22 ; Davidson, viii. 248 ; Bally, p. 215 ; Towne, p. 21 ; Wragg, x. 74 ; Gillespie, p. 38 ; Dickinson, p. 126 ; Savarésy, p. 269 ; Arnold, p. 8 ; O'Halloran, pp. 78, 85 ; Ticknor, iii. 224 ; T. Smith, xxxv. 42 ; Dufour, iv. 50.

² Rush, iii. 63 ; *Ib.*, iii. 208 ; Currie, p. 21 ; S. Jackson, pp. 54, 60 ; Caldwell (1805), p. 82.

³ Lining, ii. 413 ; Drysdale, i. 29 ; Chabert, p. 8 ; Harrison, ii. 131 ; Dalmas, p. 7 ; Seaman, p. 10 ; Ticknor, p. 224 ; N. O. 1839, p. 331 ; Townsend, p. 171 ; Girardin, p. 33 ; Stone, vi. 559 ; Valentin, p. 165 ; A. Smith, xxv. 501 ; Louis, pp. 242–3 ; Pym, p. 228 ; Dufour, iv. 50 ; R. Jackson (Spain), p. 73 ; Denmark, p. 299 ; De Maria, p. 72 ; Caisergues, p. 177 ; Fellowes, pp. 52, 200 ; Audouard, p. 57 ; Velasquez, p. 10 ; Pariset, p. 29 ; *Ib.*, p. 386 ; Smith, xxxv. 42 ; Rochoux, pp. 325, 476 ; Dict. des Sci. Méd., xv. 335 ; Lem-

While moist, the tongue sometimes remains, during the first stage, comparatively or quite clean, and of a pale or healthy hue.¹ It not unfrequently happens, indeed, that it retains more or less completely, especially when the disease is of short duration, its natural appearance to the last, whether the case terminate in recovery or death. This phenomenon, which was observed here in a few cases during our earlier epidemics (1798–1803), has been noticed more or less frequently elsewhere, and is pointed out by Dr. Gillkrest, whose attention was called to it both in the West Indies and at Gibraltar.² More generally the tongue, during the first period of the disease, is coated with a white cotton and velvet like mucus, which in some instances is in streaks, but more frequently extends over the whole surface; while in others it leaves the edges, or tip, or both, uncovered.³ In some instances, this coating is somewhat thick, and, as it were, firm; but, on other occasions, it is of so delicate a texture as to impart to the organ the appearance of being covered with a piece of fine muslin or gauze, or of being smeared over with milk and water. Less frequently—sometimes from the onset, at others after the white coating just mentioned has existed for a time, and usually on the accession of nausea—the mucous covering of the tongue assumes a light buff, yellowish, or

priere, ii. 65; Hillary, p. 151; R. Jackson, i. 66; Dariste, p. 159; Bally, p. 214; Morgan, iv. 4; Comrie, xiii. 170; Madrid, p. 5; Peixotto, i. 412; Evans, p. 244; Copland, iii. 154; Osgood, p. 11; Grant, p. 32; Rouppe, pp. 305–308; Jolivet, p. 8; Lefoulon, p. 67.

¹ Caldwell, p. 82; Waring, p. 46; Hill, v. 90; Irvine, p. 28; Stone, ii. 559; Drysdale, i. 29; Bayley, p. 93; Barton, p. 9; Archer, v. 67; Barrington, pp. 12, 311; Gillkrest, ii. 270; Smith, xxxv. 42; Fellowes, pp. 52, 200; Pym, p. 234; Rufz, p. 11; R. Jackson, Sp., p. 82; A. Smith, xxv. 501; Savarésy, pp. 279, 280; New Orleans in 1820, p. 8; Frost, pp. 13, 33; Copland, iii. 140.

² McArthur, pp. 346–348; J. Clark, p. 7; New Orleans in 1820, p. 8; Barton, p. 9; Ralph, ii. 65; Joubert, p. 966; Louis, p. 243; Rufz, p. 11; Velasquez, p. 13; Rochoux, pp. 327, 476; Kelly, xiv. 377; Wragg, x. 74; Harrison, ii. 133–136; Imray, liii. 79; Stone, vi. 559; J. Warren, p. 502; Savarésy, pp. 279, 280; Waring, p. 46; A. Smith, xxv. 502; T. Smith, xxxv. 42; Copland, iii. 140; Randolph, xxiii. 168.

³ Rush, iii. 63, 208; Currie, pp. 23, 219; S. Jackson, p. 54; Addoms, p. 9; Pascalis, p. 31; Nassy, p. 21; Barton, p. 10; Davidge, p. 102; J. Warren, p. 501; Lining, ii. 413; Wragg, x. 75; Shecut, p. 119; Drysdale, i. 29; Seaman (1795), p. 10; Monson, p. 179; Tully, p. 296; Bayley, p. 93; C. Drake, xxi. 134; Valentin, p. 165; Gros, p. 9; Ticknor, iii. 223, 224; E. H. Smith, p. 124; Townsend, p. 171; Kelly, xiv. 378; Thomas, p. 83; Harrison, ii. 133; Dalmas, p. 7; Chabert, p. 8; T. Smith, xxxv. 42; O'Halloran, p. 78; Nott, Charleston Journ., iii. 10; Pariset, pp. 29, 386; R. Jackson, Spain, p. 70; Denmark, p. 299; Louis, p. 242; Gilpin, v. 322; Audouard, p. 57; Velasquez, p. 10; O'Halloran, p. 125; Caisergues (Italy), p. 177; Jourdain, v. 257; De Maria, p. 72; Wilson, p. 9; Belcher, xxiii. 249; Gilbert, p. 65; Dariste, p. 159; Imray, liii. 79; Bally, p. 214; Moseley, p. 436; Hunter, p. 66; Chisholm, i. 164; R. Jackson, i. 66, 105; Rouppe, pp. 305–308; Bancroft, p. 9; Osgood, p. 11; Pugnet, p. 355; Ralph, ii. 68; McArthur, p. 346; Comrie, xiii. 170; Madrid, p. 5; Rufz, p. 11; Frost, xiii. 29–33; Peixotto, i. 412; Bruce, p. 278; Fontana, p. 73; Vatable, p. 345; Evans, p. 244; Holliday, p. 8; Diet. des Sci. Méd., xv. 335; Dickinson, p. 126; Blane, p. 440; Rochoux, pp. 325, 476; Barry, in Boyle, p. 271; Grant, p. 32; Jolivet, p. 8; Vincent, p. 23; Lefoulon, p. 67; Mabit, p. 10; Bone, p. 4; Anderson, p. 5; Lallemand, p. 95.

brownish hue;¹ or it presents a pale green (*Ralph*, ii. 65–68), or an ash colour (*Archer*, v. 67). In some cases the yellow coat is spread all over, in others it appears in streaks, one running on each side of the tongue, nearly to the tip—the centre being clean or brown.

In some instances, while the tongue is coated in the way mentioned, the membrane at the edges and tip retains its natural hue, or is even paler than in health; but more commonly it presents a red appearance of greater or less intensity;² and though sometimes larger, swollen, and even flabby,³ is often pointed and contracted.⁴

The tongue sometimes continues in this condition—moist, stained white, yellow, or brown, and with red edges—until death, when this takes place on the fourth, fifth, or sixth day.⁵ Still more frequently it remains unchanged till the close, when the case ends favourably, especially when the disease has not assumed its malignant garb, and has been prolonged beyond the fifth or seventh day. In other instances, however, changes take place in the condition of the organ during the progress of the disease—sometimes in cases which ultimately terminate in a favourable way, but more particularly in those that have a fatal tendency. In such instances, after presenting, during the period of remission or metaptosis, a less unhealthful appearance than it had done before, losing some of its redness, acquiring more moisture, and showing a disposition to clean, the tongue, on the accession of the third stage, becomes more thickly coated than it was before, and rapidly assumes a highly diseased aspect. In some, it becomes foul, heavily loaded with sordes, especially towards the base, and dry; sometimes with clean, moist, red,

¹ Gros, p. 9; New Orleans in 1820, p. 8; Dalmas, p. 7; C. Drake, xxi. 134; Chabert, p. 8; Thomas, p. 38; Waring, p. 46; New Orleans in 1839, p. 332; Hill, v. 90; Rush, iii. 208; Caldwell (1805), p. 82; Monson, p. 179; Seaman, p. 10; A. Hosack, p. 14; New Orleans in 1819, p. 8; Tully, p. 296; E. H. Smith, p. 124; Townsend, p. 171; Kelly, xiv. 377; Archer, v. 67; Comrie, xiii. 170; Caillot, p. 16; Morgan, iv. 4; Osgood, p. 11; Audouard, p. 57; Evans, p. 244; Levacher, p. 71; Rouppe, p. 305; Rochoux, pp. 325, 476; Moseley, p. 437; Chisholm, i. 164; Wilson, p. 9; Imray, liii. 79; R. Jackson, i. 66; Dyott, p. 1003; Levacher, p. 71; Gillespie, p. 38; Davidge, p. 102; Frost, xiii. 29–31; Pariset, p. 29; Lefoulon, p. 371; Finlay, p. 14; Jourdain, v. 258.

² Nassy, p. 21; Addoms, p. 10; New Orleans in 1820, p. 9; Pascalis, Feb. of 1797, p. 31; New Orleans in 1819, p. 8; Waring, p. 46; Dalmas, p. 7; A. Smith, xxv. 501; She-cut, p. 119; Gros, p. 9; Stone, vi. 559; Barton, xxi. 9, 10; Townsend, p. 171; Kelly, xiv. 377; Girardin, p. 33; Thomas, p. 38; Harrison, ii. 131; Merrill, ix. 244; Ticknor, iii. 244; Pariset, p. 386; Louis, p. 242; Audouard, p. 57; Velasquez, p. 10; Gillkrest, ii. 270; Chisholm, i. 164; Dariste, p. 159; Bally, p. 214; Belcher, xxiii. 242; Rufz, pp. 11–14; Pugnet, p. 355; Vatable, p. 345; Evans, p. 244; Rochoux, pp. 326, 476; Arnold, p. 8; Levacher, p. 71; Jolivet, p. 8; Vincent, p. 24; Blair, p. 64; Lefoulon, p. 67; Copland, iii. 141.

³ Caisergues, p. 177; R. Jackson, Spain, p. 74; Gillkrest, ii. 270; Kelly, xiv. 377; Townsend, p. 145; Audouard, p. 17; New Orleans in 1820, p. 8; A. Smith, xxv. 501.

⁴ Townsend, p. 145; New Orleans in 1820, p. 9; Archer, p. 67; Girardin, pp. 33–55; Harrison, ii. 131; Pugnet, p. 355; Audouard, p. 57.

⁵ Rochoux, pp. 327, 477; Bally, p. 215; Rouppe, p. 305; Chisholm, i. 164; Nassy, p. 21; Hillary, p. 151.

or livid margins, while the papillæ enlarge, and contribute in imparting to it a rough feel and a chapped appearance.¹ It not unfrequently happens that in this stage of the disease the fur on the tongue presents yellow, brown, dark, or black longitudinal stripes, or a band in the centre. This band is sometimes dry, at others it is moist, and in many instances it leaves the edges moist, clean, white, or red.² In some cases there exist two, three, or even four of these longitudinal bands. Though often of a smooth yellowish-brown colour, remarkably glazy in appearance, they at times present a very different appearance; the one may be white and mucous, and the second red and smooth, while a third is of a dark brown colour and clammy; from one blood may exude, while the others are free from such effects. Pariset, who has noticed these phenomena, states that in some cases the hemorrhage takes place from one-half of the tongue, the other half remaining moist, clean, and slightly mucous; sometimes one-half is dry, and the rest is moist (p. 415). Dr. O'Halloran informs us that, in a case which fell under his observation, one-half of the tongue was blue, as if approaching to gangrene, the other half red. The individual recovered (p. 125).

In many cases, by the enlargement of the brown bands above mentioned, or without the previous existence of these, the whole surface assumes a dark brown or black colour, which is generally, though not necessarily, associated with dryness and roughness, and contraction of the organ, and imparts often the idea of its having been seared with a hot iron, or rubbed over with lunar caustic.³ This condition of the tongue often continues with little or no change to the close of the disease; but in another set of cases the fur, at this period, clears off more or less completely, leaving the surface clean, smooth, glossy, and of a fiery red, or livid colour—with or without dryness—or rough from swelling of the papillæ. A livid tongue, moist, thick, and red—continuing free from slime or fur till a late period of the disease, and then grow-

¹ S. Jackson, pp. 54-60; Rush, iii. 208; Addoms, p. 10; Currie, p. 24; Tully, p. 296; Ticknor, iii. 227; Townsend, p. 149; Seaman, p. 10; Bayley, p. 94; Moultrie, p. 7; New Orleans in 1839, p. 333; Wragg, x. 75; Holliday, p. 10; Arnold, p. 8; Moseley, p. 438; Hunter, p. 66; Hillary, p. 151; Caillot, p. 19; Maher, p. 842; Frost, xiii. 30; Imray, liii. 79; Savarésy, p. 275; Fontana, p. 73; Pugnet, p. 355; Gillespie, p. 38; Levacher, p. 73; Bruce, p. 278; R. Jackson, Spain, pp. 71, 82, 100; Louis, p. 242; Audouard, p. 60; Berthe, p. 85; Caisergues, p. 168; T. Smith, xxxv. 42; Denmark, p. 300; Rochoux, pp. 276, 326; O'Halloran, p. 125; Jolivet, p. 10; Vincent, p. 24.

² Rush, iii. 63; S. Jackson, p. 54; Townsend, p. 149; Archer, v. 67; Kelly, xiv. 378; Rochoux, p. 476; Madrid, p. 5; Bruce, p. 278; Velasquez, p. 11; Pariset, p. 30; Caisergues, p. 165; Berthe, p. 85.

³ Rush, iii. 63; A. Hosack, p. 15; Valentin, p. 165; Seaman, p. 10; Bayley, p. 94; Archer, v. 67; E. H. Smith, p. 124; Kelly, xiv. 377; Chisholm, i. 164; Frost, xiii. 31, 34; R. Jackson, i. 87; Wilson, p. 9; Amiel, p. 269; Poissonnière, p. 50; Heustis, pp. 111, 112; Madrid, p. 26; Savarésy, pp. 274, 277, 8; Barrington, xii. 312; Bancroft, p. 13; Hunter, p. 66; Caillot, p. 18; Evans, p. 245; Lempriere, ii. 84; Grant, p. 32; Bruce, p. 278; Barry, p. 271; Holliday, p. 10; Dict. des Sci. Méd., xv. 335; Dickinson, p. 131; Velasquez, p. 11; Arnold, p. 8; Moseley, p. 438; Gillespie, p. 78; Blin, p. 6; Fellowes, p. 292; Pariset, p. 30, ib. 414; Audouard, p. 62; Gillkrest, ii. 271; Berthe, p. 85.

ing brown and dry towards the base—has been described ; but is of rare occurrence¹ (*Townsend*, p. 145).

While such are the usual appearances of the tongue in the various stages of the disease, the organ is not unfrequently affected with tremulousness, which is sometimes carried so far as to produce difficulty of articulation. This symptom occasionally occurs early in the disease ; but much more frequently it makes its appearance at an advanced period.²

It is often combined with a clean, moist, and swollen condition of the organ. Dr. Wragg, who, like some others, has noticed it, remarks very properly, that it would be difficult for any one, not conversant with yellow fever, to distinguish this tongue from that of the habitual inebriate, and that if the watery eye, the nervous pulse, the stupid face, and stammering speech did not suffice to complete the delusion, the observer must be gifted with uncommon perspicacity.³

We have no means from published records of ascertaining the relative frequency with which the various symptoms presented by the tongue were met with in the yellow fever of this city, or, indeed, in most other places. The following table will convey an idea of the occurrences in that respect in the Roper Hospital, Charleston, during the sickly season of 1854, and is used under the impression that the results there obtained correspond pretty accurately, under ordinary circumstances, with those noticed in other places. It must be remarked that the same tongue gave many different symptoms.

The tongue in the	1ST STAGE.	2D STAGE.	3D STAGE.	TOTAL.
Was swollen	4	29	31	64
“ dry	52	23	14	89
“ bloody	00	3	31	34
“ whitish	44	33	18	95
“ brownish	94	53	39	186
“ moist	109	109	110	328
“ red	43	26	33	102
“ velvety and white . . .	23	2	3	28
“ black	1	1	19	21
“ natural	26	10	7	43
“ glazed	4	2	00	6
“ cracked	00	3	00	3
Total number of observations				999

¹ Rush, iii. 63 ; Tully, p. 296 ; Deveze, p. 23 ; Hill, v. 90 ; Valentin, p. 165 ; Townsend, p. 158 ; R. Jackson, i. 70 ; Imray, liii. 79 ; Bally, p. 215 ; Ralph, ii. 68 ; Gillespie, pp. 42, 3 ; Blair, p. 65 ; Rochoux, p. 327 ; Fellowes, p. 56 ; Pariset, p. 414 ; R. Jackson (Spain), p. 100 ; Audouard, p. 58 ; Pariset, Obs., p. 30 ; Lempriere, ii. 65 ; Boyle, pp. 230, 290 ; Velasquez, p. 12 ; Chabert, p. 8 ; McArthur, p. 348 ; Copland, iii. 155 ; Frost, xiii. 34.

² N. O., 1839, p. 333 ; Girardin, p. 55 ; Wragg, x. 74 ; Pariset, p. 30 ; Amiel, p. 263 ; Denmark, vi. 300 ; Fellowes, p. 202 ; Rochoux, p. 327 ; Dickinson, p. 131 ; Rufz, p. 15 ; Barry (in Boyle), p. 271 ; Louis, p. 242 ; Caldwell, p. 82 ; Imray, liii. 81 ; Gillkrest, ii. 270 ; Rouppe, p. 307.

³ Charleston Journ., x. 75.

Thus, as is remarked by the distinguished gentleman from whom the above is borrowed, it will be seen that the tongue was moist in the greater number of cases, and, what is particularly worthy of note, it was so in the three stages, and in about the same absolute numbers in each stage. This indicates further, that the cases which commenced with moist tongue preserved this state to the last. The peculiarity next in point of frequency was the brownish fur. This was usually found along with the moisture, but did not, like it, continue throughout the other stages. It cleared away in those cases in which convalescence was setting in, and gave place in the more violent instances to a black, hemorrhagic, glazed, or cracked condition. The red tongue was next in frequency, being most common in the first and third stages. In the first stage, it was coincident with tenderness of the epigastrium, and was always the precursor of serious gastric trouble. The whitish tongue was next in frequency. It was met with in those cases where the fever was ushered in with rather a mild train of symptoms, but was not always without danger, as some of the most insidious cases originated so. The dry tongue came next, and after it, the swollen. These symptoms generally went together, but not always; for often the swollen tongue was moist, giving occasion to the term flabby, which has been used to distinguish these cases. The swollen tongue was always recognized as a dangerous symptom, for when accompanied with moisture, it announced the advent of serious gastric disorder, and when dry, glazed, and cracked, the approach of a no less dangerous train of nervous derangement. The tongue was natural in 43 cases, in 7 of which it continued so to the 3d stage. In 34 it was bloody, 31 being in the 3d stage; and, it is important to remark, that in many of these the coming on of hemorrhage was synchronous with the setting in of convalescence.¹

But although, as already stated, the picture here presented of the various appearances of the tongue may apply, under ordinary circumstances, to occurrences elsewhere, yet those appearances vary somewhat in point of frequency and extent in different epidemic seasons, whether in different or the same places. At some periods the tongue is more commonly moist, and so continues longer than at others. The same observation has been made in relation to the white or yellow stain of the early stage. The black, furrowed, and chapped tongue, which was very frequently noticed in most of our epidemics, was seldom if ever seen by Mr. Rochoux during a residence of several years at Guadaloupe. The clean and red tongue is not a usual symptom in some epidemics; while at Sierra Leone, in 1823 and 1829, it was encountered in all the fatal cases.² In 1794, in this city, the tongue was always moist in the commencement, but generally of a darker colour than it had been in 1793. Dr. Rush, from whom we derive this fact, remarks elsewhere that, in 1798, the tongue was not so much an index of the state of the fever as it had been in 1793 and 1797 (iii. 203). In that season it remained natural to the last in several cases attended with black vomit—a circumstance which was not noticed in the two preceding epidemics—was

¹ Wragg, *Charleston Journ.*, x. 75, 76.

² Boyle, pp. 230, 290.

seen but once in 1803, and did not occur at all in 1820.¹ Thomas mentions the coated, yellow, foul (*saburral*) tongue as occurring frequently in the early stage of the disease at New Orleans. This appearance has been seldom noticed in this city or at Natchez in 1823 or 1825. The red, polished tongue was common here in 1799, and seldom if ever met with in 1803. In 1793 and 1794 a diffused blackness of the tongue was not an uncommon symptom in the last stages of fatal cases. In 1799 and 1803, Dr. Rush saw no cases of the kind; and in 1820 they were seldom noticed, the blackness being restricted to a certain longitudinal streak, and Dr. Blane never saw a case of it (p. 440). A dark and dry tongue, such as is seen in adynamic fever, is frequent in some epidemics. In 1805 it was of very rare occurrence, and, when seen, was usually the effect of neglect or bad treatment. The same observation was made in St. Domingo in 1802-3. In 1839, at New Orleans, the tongue, during the first stage, was generally large and moist, white towards the base, not red, rarely pouted, and red on the edges and at the tip. The history of other epidemics will show that the tongue has often presented a very different appearance. In reference to the epidemic of Mobile in 1847, Dr. Nott remarks that "the perfectly clean tongue, so common in former years, was rare this season."²

The appearance of the tongue varies also according to the period of the season at which the disease is noticed. During the early part of the epidemic of 1821 at Norfolk, the tongue was generally covered with a thick, ash-coloured coat, which, about the third or fourth day, assumed a brown or black colour. In the month of October, and latter part of September, the white crust was not as often met with; but, in its stead, a brown fur appeared, and, in a few cases, it remained perfectly clean and fair until the active stage had subsided.³

Much will depend also on the character of the disease. In the inflammatory form the tongue, in the first stage, is more usually moist and whitish, or yellowish on the surface, and crimson red on the edges, and at the apex. It subsequently becomes brown, with a dark streak in the centre, swollen, moist, or clear red, bloody, black, and chapped. In the congestive form, we are more apt than in the preceding, to meet with a natural tongue, or one covered, at first, with patches of white fur, or presenting the appearance as though it had been seared with a hot iron—tremulous, dry, and fissured. In each of the forms described by Ralph, the organ presented a different appearance. In the first, it was often covered with a pale-green fur; but frequently it scarcely deviated from a natural appearance (p. 65). In the second, the tongue was frequently shining and glossy, swollen, and leaden-coloured, or covered with a thin green secretion (p. 68). In the third form, it was foul, with a thick white fur (p. 70). Similar differences are noticed in the several varieties described by Dr. R. Jackson as observed by him in the West Indies and Europe; and by every other writer who has classified, under separate heads, the various modifications assumed by the disease.

¹ Rush, iv. 41, 85; Jackson, p. 54.

² Charleston J., iii. 10.

³ Archer, Med. Recorder, v. 67.

Taking a general survey of the phenomena presented by the tongue in the yellow fever, it may, I think, be concluded that, in the early stage, the white, or light yellow stain of the organ, associated with more or less redness of the edges and apex, and more or less moisture, is more frequently observed than any other appearance, and may therefore be viewed as, to a certain extent, characteristic of the disease. The same may be said of the dark-coated and dry or semi-moist tongue, with or without dark central or lateral streaks, which are found in the latter stages; while the foul, yellow, and the dry, rough tongue of the first stage, and the foul black coat and sordes of the last are due to complications of a congestive, bilious, or adynamic kind.

b. THIRST.—Thirst is a frequent and often a troublesome symptom in the yellow fever—more so than might be anticipated, in many cases, from the nature of other phenomena which, in other diseases, are usually associated with it, and the absence of correspondence or harmony often existing between its manifestations and the condition of organs which, in other complaints, are commonly implicated when it shows itself to any extent. Moderate at the outset of the attack, it usually increases in violence as the case advances, and finally often becomes intense, especially in those instances where the perspiration is profuse. Of the epidemic of 1793, Dr. Rush says that thirst occurred in the greatest number of cases which he saw in the fever. Sometimes it was very intense. One of his patients, who suffered by an excessive draught of cold water, declared, just before he died, that he could drink up the Delaware (iii. 66). In 1794, the same physician found that a most intense degree of thirst was very common in this fever. It produced a great extravagance of expression. One of his patients went so far as to say that he could drink up, not a river, but the ocean itself (iii. 210). The frequent occurrence and urgency of that symptom at some period of the disease is also noticed by other chroniclers of our epidemics—by Deveze, who says that it is great in the first stage, and inextinguishable in the second (pp. 22, 24); by Currie, who speaks of it as considerable in the first stage, and insatiable in the next;¹ by Cathrall (p. 25), Monges (ii. 55), Nassy (p. 22). The same observation has been made in the fever of other American cities, of Europe, and of tropical regions.²

¹ *Fev. of 1793*, pp. 21, 24; *Fev. of 1797*, p. 219.

² Addoms, p. 9; Moultrie, p. 4; Archer, v. 66; Dickson, iii. 255; Cartwright, ix. 13; Hogg, *Western Journal*, i. 412, 416; Drysdale, i. 135; Barton, p. 10; Harris, xiv. 46; Tully, p. 296; A. Smith, xxv. 541; Bayley, p. 96; Townsend, p. 160; Pariset (*Obs.*), p. 19; Velasquez, p. 11; Proudfoot, xxvii. 250; Heustis, p. 12; Hayne, *Charleston J.*, vii. 10; Waring, p. 46; Stone, vi. 560; E. H. Smith, p. 124; Ticknor, iii. 225; N. O. 1839, pp. 331, 333; Barrington, pp. 311–12; Dufour, iv. 51; Caisergues, p. 177; Berthe, p. 86; Louis, p. 168; S. Jackson, p. 71; Dyott, p. 1003; Belcher, xxiii. 250; Maher, p. 848; Evans, p. 256; Lempriere, ii. 65; Holliday, p. 8; *Dict. des Sci. Méd.*, xv. 335; Imray, liii. 79, lxiv. 319; Rochoux, pp. 323–4; Wilson, p. 9; R. Jackson, *Tr.*, p. 254; Gillespie, p. 38; Poissonnière, p. 50; Moseley, p. 436; Ralph, ii. 65, 68; Comrie, xiii. 170; Frost, xiii. 29; Osgood, p. 9; R. Jackson, *Sketch*, i. 66, 68; Hunter, p. 66; Hillary, pp. 149, 151; Pugnet, pp. 353, 355; Vatable, p. 345; Arnold, p. 18; Towne, p. 21; Jolivet, p. 8; Mabit, p. 10; Dupont, p. 18; Bertaud, p. 12; Joubert, p. 966.

But while the yellow fever is often characterized by as great a degree of thirst as other diseases of a febrile character—while, indeed, in many cases, that symptom attains an excess seldom encountered in kindred affections, and does so under circumstances when it might be least expected—instances occur in every epidemic, sometimes in a large number, at others in great majority, in which thirst is moderate, or far from being distressing. This often occurs, although other symptoms present themselves at the same time of a nature calculated to make us suppose that this symptom would manifest itself also. Nor is it uncommon to find the disease differing still more widely from other fevers by being unattended with any thirst, and even by exhibiting a disrelish for drink. Many, if not most of the writers above mentioned, have recorded such facts, for few regard great or intense thirst as the invariable attendant on this disease. In this city, Dr. Rush tells us, in reference to the fever of 1793, that the thirst was moderate or absent in some cases (p. 66). In 1794, he saw some in whom it was equally absent, though the skin was hot, and the pulse quick. Nassy found that some patients were not thirsty though their tongues were dry (p. 23). Caldwell found, in 1805, that patients seldom suffered much from thirst (p. 81). Similar observations were made in 1820 and 1853. In New York, in 1795, Dr. Bayley, while stating that, in some instances, it was difficult to satisfy the demands of the sick for drink, remarks, nevertheless, that “although the fever frequently ran high, it was seldom attended with much thirst” (p. 96). Dr. Hosack, of the same city, states, as the result of his experience, that there is no thirst, or that it is not troublesome.¹ Dr. Monson, of New Haven, found it very inconsiderable (p. 179). Dr. C. Drake² found it moderate in 1819. Lining, speaking of the fever of Charleston in 1748, says that, in the first stage, “the thirst in very few was great;” and that in the subsequent one “very few complained of thirst, though they had a great desire for cold liquors” (ii. 413, 419). Dr. Dickson, though stating that, in 1817, the thirst was excessive, expresses his belief that “water was as often desired on account of the agreeable sense of coolness which it produced in the burning stomach, as for the relief it gave by quenching thirst” (iii. 255).

In New Orleans, thirst would appear to be as often absent as present, and generally moderate, as we learn from the statement of some writers;³ and as may be inferred from the silence of Gros, Girardin, Harrison, &c., on the subject.

Dr. Drysdale states that, in Baltimore, in 1794, it was generally moderate, and in some cases absent (i. 135). Nearly similar observations are recorded by Dr. A. Smith, of Galveston (xxv. 501).

In Europe, during the epidemics of Cadiz, Xeres, Barcelona, Port de Passage, Seville, Gibraltar, &c.,⁴ thirst, so far from being a prominent

¹ Practice, p. 388.

² Med. Repos., xxi. 134.

³ N. O. 1839, p. 331; Chabert, p. 9.

⁴ Pariset, p. 387; Rochoux, p. 475; Audouard, p. 56, &c.; R. Jackson, *Fev. of Spain*, pp. 80, 95; O'Halloran, pp. 79, 126; Pym, pp. 228, 229, 234; Jourdain, v. 257–8; De Maria, p. 72; Bahi, p. 9; Copland, iii. p. 140.

symptom, was often absent and generally moderate ; while, in tropical climates, the fever is not unfrequently marked by a similar absence, or moderate degree of that symptom. Not a few writers—as Desportes, Bancroft, Warren—take no notice of it at all ; and others mention it incidentally, and in terms indicating its not having attracted special attention. According to Blane (p. 440), “the state of the fauces is different from that of most other fevers, *for there is no excessive thirst.*” Hillary (p. 149), Hunter (p. 66), Wilson (p. 182), Dariste (p. 159), Rufz (pp. 11, 13), Fontana (p. 73), testify as to its being as often moderate as otherwise. Dr. J. Clark states that, at Dominica, “the sick had not much desire for drink” (p. 7). Bally (pp. 216, 239) found it almost invariably very moderate. Dr. R. Jackson, in his treatise on the diseases of Jamaica, states that the thirst in true yellow fever was seldom great—often “very little increased beyond what it naturally is” (pp. 254–8); and in a later publication, he says: “The sensation of thirst is sometimes very conspicuous, sometimes little troublesome, in those who are under the influence of febrile action” (*Sk.* i. 168). Chisholm, in his account of the epidemic of Grenada, remarks that “the thirst was not very considerable in general” (i. 174); according to Bourdon, “in all cases thirst is moderate” (p. 9). Rufz says of the opening stage: “There was more frequently disgust than desire for drinks ;” and, in speaking of the next period, he adds: “If there had been thirst, it now diminishes” (pp. 11–13). Lefoulon states that, though sometimes intense, thirst is usually scarcely felt (pp. 67, 371).

The prevalence of thirst, and the extent it attains, appear to differ at different periods. It was more generally felt, in this city, in 1793, '94, than in 1805, 1820, and 1853 ; while Chisholm found it inconsiderable at Grenada in 1793, Rochoux came to different results at Guadaloupe ; Holliday, at the Havana in 1794 ; Imray, at Dominica in 1838.

Much also will depend on the form which the disease assumes. It is, as a general rule, much more marked in the inflammatory than in the congestive and adynamic forms of the disease, seldom completely absent in the former, frequently so in the latter.¹ It will be found, moreover, that the form of the disease being the same, thirst is more urgent in cases of fatal tendency than in those that end favourably. It is generally greater during the period of reaction—and diminishes or disappears during the calm of the remission, often to return—sometimes in a moderate, at others, in an intense degree—during the latter stages. The sensation of thirst is often connected with a state of dryness and redness of the tongue and lips. But, unlike what takes place in other febrile affections, thirst is sometimes insatiable where the tongue deviates little, in appearance, from its natural state ; and, on the contrary, “in some instances, the tongue is rough and dry as a potsherd, without a distinct sensation of thirst.”²

c. RESPIRATION.—The condition of the respiratory function differs very considerably not only in different individuals during the same year, but, as it would seem, in different epidemic seasons. In some instances, it is performed

¹ R. Jackson, Wilson, Pym.

² R. Jackson, i. 168.

during the whole of the first stage with the utmost freedom and ease, and in others the breathing is much less embarrassed than it is in ordinary fevers—less than might have been anticipated, from the tumult under which the rest of the functions are at the time labouring. The absence of respiratory derangements in the yellow fever, a condition which might be inferred from the silence of many writers on the subject at this period of the disease,¹ is pointed out as a usual or occasional occurrence, not in temperate regions alone, as Dr. Rochoux would endeavour to persuade us, but in tropical climates too, by several competent authorities,² and has been observed in the epidemics of this and neighbouring cities. Nor is it less true, that in some seasons this entire or comparative freedom from the derangement in question has often continued unchanged during the whole course of the disease. Such we may presume was the case at Barcelona in 1821, not only from the positive statement of Bally (p. 518), and the silence as to the state of the respiration on that occasion on the part of Audouard and others, but from the fact that Rochoux, who contends for the immunity in the yellow fever of temperate regions generally, derived his knowledge of the phenomena of that disease from what he saw during that memorable epidemic. Such is also stated, though with doubtful correctness, to have been the case in St. Domingo in 1802 (*Bally*, p. 220); at Carthage in 1804 (*Ib.*, p. 267), and at some other periods.

Much more frequently, respiration during the stage of reaction is more or less affected—the change from the standard of health varying in different individuals. The patient complains of oppression. His breathing is difficult, quick, or slow, short, laborious, interrupted, asthmatic, panting, gasping. He takes long inspirations, and complains occasionally of a sense of suffocation or strangulation. Our several epidemics have afforded many instances of those various modes of derangement.³ Speaking of the epidemic of 1820, Dr. S. Jackson says: “The respiration in some was very laborious and hurried; in others it was slow; some suffered from a feeling of suffocation which was ascribed to an impossibility of inflating the lungs, and was accompanied with violent spasmodic pains of the chest” (pp. 55–6). Similar has been the result of observations made in other parts of this country as well as in Europe and tropical regions; in all of which the state of the respiration has been described in terms analogous to those I have made use of.⁴ If some writers merely state that it is hurried or slow, or interrupted

¹ Bruce, Lempriere, A. Drake, Vatable, Bancroft, Dufour, Madrid, Copland, McArthur, Louis, Harrison, Davidson, Imray, A. Smith.

² Pariset, p. 393; Rochoux, p. 512; Ralph, ii. 75; N. O. 1839, p. 333; Dariste, p. 160; Bayley, p. 97; Bally, p. 220; Caldwell, p. 81; Stone, vi. 555; Rufz, p. 13; Lining, ii. 413; Wragg, x. 74.

³ Deveze, p. 22; Currie, pp. 21, 218; Cathrall, p. 23; Barnwell, p. 369; Caldwell (1805), p. 81; Ffirth, p. 27.

⁴ Chabert, p. 8; Moultrie, p. 3; Addoms, p. 9; Townsend, p. 146; Heustis, p. 110; Bayley, p. 97; Gros, p. 9; Valentin, p. 166; Waring, p. 46; Hosack, Practice, p. 388; Warren (in Tytler), p. 501; Thomas, p. 31; Dickson, iii. 254; Girardin, p. 33; New

or laborious—others, as Dr. R. Jackson, affirm that, in the West Indies, “gasping for breath, or an unceasing attempt to fill the lungs without power to do it, is not uncommon.”¹

During the period of remission, the respiration, when it has previously been disturbed, usually resumes its pristine condition—becoming slow, regular, easy. At other times, however, when the remission is not complete, it continues or becomes slower or quicker, weaker than natural, *suspirious*, or otherwise disturbed. In the subsequent stage, even in cases in which it was, at first, quite or nearly free, the function becomes usually considerably—often painfully—affected, and continues so to the close of the disease. It is difficult, stertorous, laborious, suffocating, deep, panting, heaving, convulsive, irregular—sometimes characterized by deep inspirations, or by long expirations, followed by short inspirations; in some cases, it is hurried and fluttering; in many, it is slow; this slowness becoming greater and greater as the disease advances. Such conditions of the respiratory function attain, at times, a distressing degree of aggravation, and prove a source of insufferable distress to the patient. They have all been observed, more or less frequently, in this city and elsewhere, and are described not only by many of the writers already mentioned; but by others who take no notice of the state of the function in the preceding stages, or represent it as deviating little, if at all, from the standard of health.² The same may be said of those more marked cases of the congestive kind in which the disease consists, as it were, only of the stage of prostration; or in which the preceding stage of reaction has been transient and imperfect; for in these the laborious, suffocating, panting respiration is often encountered,³ even from the commencement of the attack.

Orleans 1820, p. 8; A. Hosack, p. 12; Randolph, Med. Repos., xxiii. 168; Pascalis, v. 143; Archer, v. 66; Barrington, xii. 311; Drysdale, i. 126; New Orleans, 1839, p. 332; O'Halloran, p. 81; Berthe, p. 82; Caisergues, p. 168; Velasquez, p. 10; Pariset, Obs., p. 30; Gillkrest, ii. 271; Louis, p. 244; Palloni, p. 4; Jourdain, v. 257, 8; Arejula, p. 160; Shott, p. 26; Hume, p. 201; Dict. des Sci. Méd., xv. 335; Hillary, p. 147; Gilbert, p. 65; R. Jackson, i. 67, 87, 71, 165; Poissonnière, pp. 7, 50; Dariste, p. 160; Moseley, p. 436; Holliday, p. 9; Osgood, p. 10; Towne, p. 21; Savarésy, pp. 269, 272, 280; R. Jackson, Tr., pp. 254, 274; Hunter, p. 64; Dickinson, pp. 126, 7; Pugnet, p. 353; Gillespie, p. 38; Evans, p. 256; Warren, p. 10; Ralph, ii. 65, 8; Frost, xiii. 29; Proudfoot, xxvii. 249; Vincent, p. 23; Mabit, p. 10; Jolivet, p. 8; Berthaud, p. 10; Leblond, p. 103; Joubert, p. 966; Bone, p. 5; Lallemant, p. 84; Grant, 30; Heastie, p. 20; Carter, p. 4; Anderson, p. 5.

¹ It is to be remarked, that the statement of Bally that in St. Domingo the respiration was free, not in the first only, but also in the advanced stages of the disease, is at variance with the statements of Gilbert, Vincent, Mabit and others, who witnessed the same epidemic, and also with the details of the cases he himself described; for, in forty cases—the whole number being ninety—respiration is mentioned in seventeen. In fourteen of these, it is stated to have been variously affected. In one out of the remaining three, there was an affection of the brain which concealed the condition of the respiratory function, and retarded it.

² Bruce, p. 279; Vatable, p. 346; Rufz, p. 15; Madrid, p. 6; Ib., pt. iii. 31; McArthur, p. 347; Lempriere, ii. 64; Copland, iii. 158; Louis, p. 244; C. Drake, xxi. 135; Defour, iv. 51; Lining, ii. 425; Bancroft, p. 15; Pariset, p. 425.

³ Merrill, ii. 223; Ib., ix. 245; R. Jackson, i. 91.

While respiration presents the aforesaid evidences of greater or less derangement in the several stages of the disease, it is very generally interrupted at an early, though more particularly at a later period, by deep sighs.¹ At the same time, the expired air in the stage of reaction is hot and burning, and often cold in the last; while, at an advanced stage of the disease particularly, the breath, as already seen, is offensive—the odour being of a peculiar acid alliaceous character. In some epidemics this is so frequently encountered as to have been termed “the yellow fever odour,” and is regarded as among the characteristics of the disease.² This odour must be distinguished from that exhaled from the cutaneous surface, and must not be confounded with that resulting from the decomposition of blood in the mouth. It is *sui generis*, resembles no other odour, and an idea of it can only be formed by actual experience. It is not always present within the first twenty-four hours after the attack, and sometimes is not observed at all, or is so faint as to easily pass unheeded. In some epidemics, indeed, it is said to have not presented itself in any case, though the greatest care was taken to ascertain the fact.³ On the other hand, it shows itself very generally in some places, is discovered early in the disease, and may, by an experienced observer, be detected even before the onset of the attack. “During my residence in the Havana (1839)”, says M. Bertulus, “I had frequent opportunities of conferring with M. Belot, a French practitioner who has resided in that city for more than thirty years. On one of these occasions he spoke to me of the symptom in question; and, though at first incredulous on that point, I soon became convinced of the correctness of his observations. I several times saw this able practitioner predict an attack of yellow fever to sailors of my ship—and that, too, six or eight days before the accession of the first symptoms.” M. Bertulus himself was warned by M. Belot of the danger to which he was exposed. In this case the prediction was verified.⁴

In addition to the pulmonary derangement, evinced by disturbed respiration, the patient is sometimes troubled with a slight hacking and nervous

¹ Girardin, p. 33; Dickson, iii. 254–256; Merrill, ix. 244; S. Jackson, p. 55; Lining, ii. 425; Pascalis, v. 143; Dalmas, p. 10; Rush, iii. 52; Caisergues, p. 168; Dickinson, p. 126; Pariset, p. 425; Ralph, ii. 67–70; Grant, p. 34; New Orleans in 1820, p. 8; Berthe, p. 85; Evans, p. 256; Gillespie, pp. 40–43; Harrison, ii. 132; Peixotto, i. 413; Rochoux, pp. 291, 513; Bayley, p. 97; Bally, pp. 214–228, 328; Wilson, p. 11; Stone, vi. 553; Dariste, p. 163; James Clark, p. 16; Pascalis, p. 31; Savarésy, p. 280, 281; Pugnet, p. 353–355; R. Jackson, Tr., p. 274, ib. Sketch, i. 87–91; Moseley, p. 436; Gros, p. 9; Hillary, p. 147; Valentin, p. 166; Lempriere, ii. 64–84; Currie, p. 219; Irvine, p. 29; Fellowes, p. 202; Cathrall, pp. 23–29; Boyd, p. 300; Palloni, p. 4; Townsend, pp. 146–176; Osgood, p. 10; Vatable, p. 346; Kelly, xiv. 378; Deveze, p. 22; Dict. des Sci. Méd., xv. 335; New Orleans in 1839, p. 333.

² Rochoux, p. 514; Rufz, p. 11; Kelly, xiv. 374; Dict. des Sci. Méd., xv. 337; Audouard, p. 52; Stone, vi. 560; Pariset, p. 426; Hill, v. 90; Jolivet, p. 8; Bertaud, p. 12; Blair, p. 65; Maher, p. 873; Fever of Cayenne in 1850 and 1851, p. 175; Lalle-mant, p. 85.

³ Joubert, Fièvre Jaune, Ann. Mar., 1844, ii. 964.

⁴ Observations et Réflexions sur l'Intoxication Miasmatique, p. 35, note.

cough,¹ with more or less pain in the chest; while, on physieal exploration, we sometimes, but not often, discover a wide-spread mucous râle, loud respiratory sound, obscure crepitation, with mat sound in small detaehed points. (*Levacher*, p. 71.)

Finally, the difficulty of respiration of the advanced stages is often aceompanied with moans and shrieks, or a loud, ineessant, and monotonous wailing, extremely distressing to all within hearing.²

The hurried respiration of the early stage is apparently produaed, in some cases, by acceleration of the eirculation—keeping paec with the latter, increasing and diminishing with it. Neither it nor other derangements of that function can be referred to morbid conditions of the substanec of the lungs, which is seldom affected, even in the slight degree mentioned. More likely are all the respiratory derangements attributable to the effect upon the nervous power of the lungs, of blood contaminated by the morbid poison giving rise to the disease, and to the stasis of that fluid and congestion resulting therefrom in those organs.

d. PAIN.—Pain is an almost invariable attendant on the yellow fever. Though, like other symptoms, more universal and severe in some epidemics than in others, it is found to occur more or less at every return of the disease, and has been noted in every place where the latter has manifested itself; so constantly, indeed, as to be justly regarded as one of its most striking eharacteristics. It is generally a source of much, sometimes of the most intense, suffering to the patient. The late Dr. Rush remarks on the subject, that the sympathy of friends with the distresses of the sick extends to a small part of their misery when it does not include their sufferings from pain. One of his patients in 1793 declared, in the height of her illness, that “no one knew the pains of the yellow fever but those who felt them” (iii. 66). They are represented as excruciating and torturing, and are but too often so great as to extort groans, cries, and tears from the unhappy sufferer. In the greater number of cases, several parts are affected at the same time; while in some the pain is located in one or two only. Though sometimes equally severe in all these, it not unfrequently happens that the pain, while very acute in one part, is little or moderately felt in others; that, in some cases, while it diminishes in one part it increases in another; and that these alternations are repeated several times during the course of an attack. The pain varies, as regards its character, in different cases, as well as at different periods of the same case. It is sometimes acute, sharp, and piercing; at other times it is of a dull, obtuse charaeter: the former usually occurring in the inflammatory form of the disease, the latter in the congestive, in which sensibility is often greatly impaired. But, characteristie as this symptom may be, constantly as it may occur, and severe as it generally proves, cases occasionally present themselves in which it is scarcely, if at all, felt; and others, again, in

¹ Pugnet, p. 355; Drysdale, i. 126; Townsend, p. 154; New Orleans in 1839, p. 333; Rufz, p. 11; Boyd, p. 299; Levacher, p. 71; Rochoux, p. 292; Poissonnière, p. 7.

² Harrison, ii. 133; Gillkrest, ii. 271; Kelly, xiv. 379; Jolivet, p. 11.

which, though felt, it is of a comparatively mild character. These exceptional instances of freedom from suffering are to be found in cases in which the vitality of the system is overwhelmed by the intensely sedative operation of the morbid cause, as evinced by symptoms of prostration of the powers of life; while moderate pain is to be sought among the milder grades of the inflammatory form of the disease.

The symptom in question is the usual accompaniment of the stage of reaction. It almost invariably continues during the whole of that stage; disappears with the progress of the disease; generally ceases completely at the period of metaptosis; and sometimes, though seldom, except in parts presently to be mentioned, reappears, after a momentary respite, at a more advanced stage, when it assumes a more obtuse form, and imparts sometimes the sensation of fatigue. The pains are principally located in the head, loins, extremities, stomach, or bowels. On each some remarks must be offered.

1. *Pain in the Head*.—Pain in the head is an early and nearly constant symptom of the yellow fever. It has been observed, in a greater or less degree, in all the epidemics on record, and has attracted the notice of medical inquirers in all climates, and nowhere more than in this city, in all the sickly seasons of which it has played a conspicuous part in the catalogue of symptoms. The works of Rush (iii. 66, 209), Currie (pp. 19–21, 218), Deveze (pp. 21–37), Barnwell (pp. 369–372), S. Jackson (p. 52), Caldwell (1805, p. 81), Nassy (p. 20), Pesealis (p. 30), Cathrall (p. 24), and Ffirth (p. 26) afford the most ample proofs on that score; while the numerous writings we possess on the fever of other parts of the United States,¹ of Europe,² and of tropical climates,³ establish beyond dispute the universality

¹ Drysdale, i. 127; Dalmas, p. 7; Gros, p. 9; Dickson, i. 348; Girardin, pp. 33–55; Barton, pp. 9, 10; Cartwright, ix. 13; Lining, ii. 411, 414; Bayley, p. 93; Randolph, xxiii. 167; Merrill, ix. 244, 245; Dickson, iii. 253; Ticknor, iii. 223, 224; Townsend, p. 144; Archer, v. 66; Hill, v. 89; Tully, p. 295; C. Drake, xxi. 134; Valentin, p. 164; Baxter, xxi. 3; Nott, Charleston Journ., iii. 9; Revere, iii. 224; J. Warren, in Tytler, p. 501; Thomas, p. 83; Brown, p. 14; Kelly, xiv. 377; Shecut, p. 119; New Orleans in 1819, p. 8; Ib. in 1820, p. 8; A. Hosack, p. 11; Moultrie, p. 5; Harrison, ii. 133; Seaman, iv. 249; Stone, vi. 556; Waring, p. 46; Wragg, x. 72.

² Proudfoot, xxvii. 249; Caisergues, pp. 165, 166; Berthe, pp. 77–81; Smith, xxxv. 42; Jourdain, v. 257, 258; Amiel, p. 363; Boyd, in Johnson, p. 299; Blin, p. 6; Velasquez, p. 9; Pariset, Obs., p. 29; Gillkrest, ii. 270; Arejula, p. 159; Palloni, p. 4; Rochoux, p. 484; Pym, pp. 228, 229; Pariset, pp. 382, 383; Dufour, iv. 50; R. Jackson (Spain), pp. 70, 95; Lafuente, in Burnett, p. 490; O'Halloran, p. 77; De Maria, p. 67; Fellowes, pp. 52, 200; Louis, p. 177; Audouard, p. 56.

³ Rochoux, p. 299; Savarésy, pp. 269–271; Imray, liii. 79; Bancroft, p. 9; Pugnet, p. 353; Lempriere, ii. 59–83; Chisholm, i. 140–160; Jackson, Tr., pp. 258–274; Fontana, p. 72; Bruce, p. 278; Warren, p. 9; Barrington, xii. 311; Poissonnière, pp. 7, 49; Ralph, ii. 65; McArthur, p. 346; Osgood, p. 9; Moseley, p. 436; Hillary, p. 147; Madrid, pp. 21–30; Blane, p. 428; Jackson, Sk., pp. 61, 65, 68, 87, 107; Desportes, i. 193; Vatable, p. 345; Peixotto, i. 412; Gillespie, p. 38; Blair, p. 64; Doughty, pp. 11, 126; Towne, p. 21; Barry, p. 271; Musgrave, ix. 117; Dyott, p. 1003; Furlong, p. 290; Belcher, xxiii. 249; Gilbert, p. 65; Frost, xiii. 29, 32; Dubreuil, viii. 321; Comrie, xiii. 169; Stewart, iii. 187; Caillot, p. 16; Davidson, viii. 248; Rufz, p. 12; Catel, p. 11;

of its occurrence. Though in some few instances—as in Baltimore in 1794, in Middletown in 1820, and in some of our own visitations—occupying the occiput, and in others the summit of the head, the pain, in by far the greater number of instances, affects the forehead and supra-orbital arches, which it embraces like a crown; as also the sockets of the eyes, and the eyeballs themselves, which feel as if endeavouring to escape (*Shecut, Bayly*). It shoots from temple to temple, with or without fulness or tightness of the eyes, and is often attended with a violent throbbing of the temporal arteries. It is usually acute, darting, piercing, and distressing—sometimes obtuse and dull. It has been compared to the repeated strokes of a hammer upon the brain; and when associated with heat, to the boiling of a pot, or to the effect of an iron circle (*Berthe*), or the binding of a cord (*Audouard, Jackson, Rush*). It has been described as “obstinate, irremovable, and distracting” (*S. Jackson*), as excruciating (*Jackson, O'Halloran, Archer, C. Drake*), torturing (*Currie, Drysdale*), exquisite (*Chisholm*), appalling (*Wragg*). A gentleman declared, in anguish, to Dr. Drysdale, that the pain through his eyes “was enough to deprive him of his senses” (p. 128); and in many cases it has caused shrieks, cries, and groans.

As may be perceived, the headache of yellow fever differs from that occurring in bilious remittents and other febrile diseases; for in these the pain more frequently occupies the upper and back parts of the head, and those parts are almost always more or less affected when the forehead suffers. We moreover but seldom observe in the latter disease that circumscribed, torturing pain in the globes or sockets of the eyes, or adjacent parts; while, on the other hand, the pain felt is more frequently attended with a sense of fulness and symptoms of cerebral congestion and irritation. In the yellow fever, the latter symptoms doubtless may, and often do, exist during the first stage, but they do not necessarily do so—and are, indeed, as often absent, though the pain is most acute and torturing. This has been observed in this country and in tropical climates, and disproves the opinion of those who refer the violent headache of the yellow fever of the latter climate to meningeal or cerebral inflammation, and deduce from this an argument in favour of a radical difference between that fever and the yellow fever of this country and Europe. Though not generally the result of meningeal or cerebral inflammation, either in the yellow fever of this country or of hot latitudes, headache of this kind, especially when it attains the degree mentioned, often seems to pre-
 sage the approach of congestion or other equally dangerous pathological condition of the brain. These, in some instances, soon show themselves, and are attended by symptoms of their own, in nowise appertaining necessarily to the disease—of which, indeed, they simply constitute complications. The violent

Dariste, p. 158; Dancer, p. 82; Bally, p. 210; Maher, pp. 842, 843; J. Clark, p. 6; Wilson, p. 8; Dict. des Sci. Méd., xv. 334; Evans, p. 256; Levacher, p. 71; Lefoulon, p. 68; Arnold, p. 8; Leblond, p. 102; Labat, ii. 2, iv. 307; Vincent, p. 23; Jolivet, p. 8; Bourdon, p. 9; Joubert, p. 966; Cayenne (1850), pp. 166–167, 174; Hume, p. 197; Grant, p. 29; Heastie, p. 18; Copland, iii. 139; Carter, pp. 3, 4; Lallemand, p. 87; Anderson, p. 4.

pain in the head is sometimes accompanied with intolerance of light; but, unexpected as the occurrence may be, in the majority of instances, even when the suffering is most excruciating—when the eyeballs are themselves the seat of pain, and the latter is aggravated by the touch or motion—there is no increased sensitiveness of those organs, or of the pain in the head, by the stimulus of light.

The pain of the head comes on with the accession of the first stage of the disease. It continues during the whole of that stage, gradually diminishes after the second day, and disappears more or less completely at the period of remission, or about the third or fourth day. It sometimes, though not often, returns after this period, and, when it does so, assumes a less acute character. The latter form is the more common attendant of the inflammatory variety of the disease, the congestive cases being frequently accompanied with a sense of stupor, weight, and oppression, rather than pain in the head; while in others the pain is of a dull, obtuse kind; and in a different set, again, there is no pain at all. But on this point, as on others, the rule is not without exceptions. Cases of the inflammatory kind—particularly those of a milder grade—present themselves, in which the pain is slight; while in some, evidently of a congestive kind, it is acute and intense.

2. *Loins*.—Not less severe, and perhaps more frequent, than the pain of the head, is that which the patient experiences in the lumbar region. Dr. Rush, in taking notice of this symptom in his account of the epidemic of 1793, remarks that it was so severe that the stoutest men complained, and even groaned, under it (iii. 65). It was noticed in that and subsequent epidemics by Currie (pp. 19–21, 218), Deveze (p. 21), Barnwell (pp. 369–372), Caldwell (p. 81), Cathrall (p. 24), Ffirth (p. 26), Nassy (p. 20), S. Jackson (p. 52), and others, and is noted elsewhere by all the writers to whom reference has been made on the subject of the pain in the head.¹ Like the latter, it is

¹ Bayley, p. 92; Moultrie, p. 6; Drysdale, i. 127; Townsend, p. 143; Baxter, xxi. 3; C. Drake, xxi. 134; Revere, iii. 224; E. H. Smith, p. 115; New Orleans in 1819, p. 8; Dalmas, p. 7; Gros, p. 9; Girardin, p. 33; Cartwright, ix. 13; Lining, ii. 411, 414; Archer, v. 66; Monson, p. 179; Waring, p. 46; Merrill, ix. 244, ii. 360; Hill, v. 89; Tully, p. 295; Valentin, p. 164; Kelly, xiv. 377; Seaman, iv. 249; A. Smith, xxv. 501; New Orleans in 1839, p. 331; Dickson, pp. 126, 127; Brown, p. 14; Harrison, ii. 131; Stone, vi. 556; A. Hosack, 12; Ticknor, pp. 222, 223; Shecut, p. 119; Barton, p. 9; J. Warren, p. 501; Thomas, p. 83; New Orleans in 1820, p. 8; Randolph, xxiii. 167; Nott, Charleston Journ., iii. 9; Dickson, i. 348; Wragg, x. 71; Fellowes, p. 52; Louis, p. 194; Audouard, p. 56; Caisergues, p. 166; Berthe, p. 81; Smith, xxxv. 42; Amiel, p. 263; Boyd, p. 294; Blin, p. 6; Velasquez, p. 9; Arejula, Edinb. Journ., i. 448; Gillkrest, ii. 270; Pariset, pp. 383, 404; Pym, pp. 229, 233; Rochoux, pp. 299, 484; Arejula, p. 159; Jourdain, v. 258; Copland, iii. 139; Savarésy, p. 279; Pugnet, p. 354; Imray, liii. 79; Lempriere, ii. 59; Chisholm, i. 149; Labat, ii. 2, iv. 307; McArthur, pp. 346, 347; Ralph, ii. 65–68; Osgood, p. 9; Moseley, p. 436; Hillary, p. 147; Madrid, pp. 21–30; Blane, p. 428; Gillespie, p. 39; Hunter, p. 64; Fontana, p. 72; R. Jackson (Spain), p. 71; Dufour, iv. 50; Bruce, p. 278; Jackson, Sketch, i. 66, 87, 104; Desportes, i. 193; Barrington, xii. 313; Dict. des Sci. Méd., xv. 334; Vatable, p. 345; Poissonnière, pp. 7–49; Peixotto, i. 412; Bally, p. 225; Maher, p. 848; Warren, p. 9; Wilson, p. 9; Frost, xiii. 29; Dubreuil, viii. 321; Comrie, xiii. 169; Steward, iii. 187; Caillot, p. 16;

often the source of intense suffering to the patient, from whom it not unfrequently extorts cries and shrieks, and has hence been justly characterized as torturing, intolerable, and excruciating. Affecting the lower part of the spine, it shoots forward towards the umbilicus, downward through the pelvis and along the thighs, and sometimes upward, along the spinal column, as high as the cervical vertebræ. In such cases, it is of a sharp, acute, lancinating character. In some instances, however, it is of a dull, obscure kind, imparting more especially the sensation of great fatigue or weight, and helpless debility about the spine, especially the sacrum, rather than actual pain; but is not the less, for that, distressing to the patient. In some instances, again, it is but slight, or altogether absent. The pain in the loins, like that in the head, comes on with the accession of the stage of reaction; and together with it—sometimes sooner, sometimes later—ceases at the period of remission; but, more frequently than the former, returns, though in a less intense degree, in the subsequent stage of the disease. Dr. Wragg, of Charleston, remarks that pain in the back was present in every single case that came into the Roper Hospital, of which he had charge, in 1854. It was one of the most distressing symptoms to the patient, and persisted, with little or no abatement, till convalescence was far advanced. “This,” he adds, “may be said to be the only symptom that was never wanting. It was intense in degree, and extended around the loins and flanks towards the lower portion of the abdomen, occasioning great distress in the space between the umbilicus and pubis” (pp. 71, 72).

Dr. Rush was of opinion that the more the pains are confined to the loins and back, the less danger was to be apprehended from the disease. Entitled as the opinion of this eminent physician may be to our regard, on questions of this kind, it will be shown, when on the subject of prognosis, that the severity of the symptom is usually proportionate to the degree of severity of the disease. Like acute pain of the head, that of the loins generally accompanies the inflammatory form of the disease; while the dull, obtuse pain, the sensation of fatigue and weight, or the absence of pain, is usually encountered in congestive cases. Exceptions to the rule are, however, more frequently met with as regards this than the other symptoms, acute pain of the loins being often an attendant on the latter form of the disease. The pain of the loins is sometimes associated with, increased or produced by, inflammation and abscess of the kidneys. But cases of most excruciating pain occur, though no such inflammation exist, and *vice versa*. It may also sometimes be due to sympathetic influence of inflammation of the stomach and bowels. But such cases, if they occur, must do so but rarely, the pain of the loins prevailing prior to the supervention of gastro-intestinal inflammation, and subsiding about the time this inflammation comes on. It is more probably

Catel, p. 11; Bourdon, p. 9; Morgan, iv. 4; Furlong, p. 290; Belcher, p. 249; Rufz, p. 13; Musgrave, p. 117; Dariste, p. 160; Lefoulon, p. 69; Joubert, p. 966; Arnold, p. 8; Gilbert, p. 65; Evans, p. 256; Barry, p. 271; J. Clark, p. 8; Dancer, p. 82; Jolivet, pp. 8, 9; Vincent, p. 23; Fever of Cayenne (1850), pp. 166, 167; Anderson, p. 4; Grant, p. 30; Carter, p. 3; Heastie, p. 28; Lallemand, p. 87.

neuralgic, and, from its severity, duration, and evident correspondence with derangements of the glandular and secretory functions of the abdominal viscera, has been regarded as located in the great sympathetic nerve.

3. *Extremities*.—We have seen that pain extends from the back downwards to the thighs. Very generally, it proceeds to the lower extremities, affecting the calves of the legs, the knees, ankles, or toes. In some cases, it affects but one of the extremities;¹ generally, it attacks both. Not less than the pains already mentioned, it is often excessively severe—assuming at times the character of muscular cramp. “One of my patients,” says Dr. Rush, “upon whose limbs the disease fell with its principal force, said that his legs felt as if they had been scraped with a sharp instrument” (iii. 66). A gentleman confessed to Dr. Drysdale, of Baltimore, that the pain in his legs was so acute “that he could cry like a child.”² Chisholm remarks, in reference to this subject: “The pain in the leg has been almost always felt immediately below the left calf, where the gastrocnemius and soleus muscles meet and form the great tendon. A considerable involuntary contraction of the leg takes place in consequence of it; and, in the point where it is chiefly felt, it communicates a sensation similar to gnawing, which, from time to time, occasions extreme torture” (i. 161). It imparts often the sensation which the French characterize by the words *brisement des os*.³ It was formerly described under the name of *coup de barre*, and has been generally designated by terms as strong as those used to denote the pains in the head and loins.⁴ At other times, it is of a vague obscure character, and assumes more the character of weariness in the limbs than actual pain. This sensation extends occasionally to the whole muscular system, imparting a feeling similar to that produced by intense fatigue. It is a source of great distress to the patient—proving often less endurable than even acute pain.

¹ Rush, iii. 209; Drysdale, i. 128.

² Med. Mus., i. 128.

³ Deveze, op. cit.

⁴ Rush, iii. 209; Deveze, p. 21; Currie, pp. 21, 218; Barnwell, p. 369; S. Jackson, p. 52; Caldwell, p. 81; Gros, p. 9; Girardin, p. 33; Dalmas, p. 7; Drysdale, i. 127; Lining, ii. 411; Merrill, ii. 222; Thomas, p. 83; Dickson, iii. 253; Essays, i. 348; Tully, p. 295; Valentin, p. 164; Baxter, xxi. 3; Stone, vi. 556; Townsend, p. 144; C. Drake, xxi. 134; Revere, iii. 224; A. Smith, xxv. 501; Seaman, p. 8; E. H. Smith, p. 15; Barton, p. 10; Monson, p. 179; N. O., 1819, p. 8; Areher, v. 66; Bayley, p. 93; Harrison, ii. 131; N. O. 1820, p. 8; J. Warren, p. 501; N. O. 1839, p. 331; Dickson, p. 125; R. Hosack, p. 11; Ticknor, iii. 222, 3; Hill, v. 89; Shecut, p. 119; Nott, Charleston Journ., iii. 9; Wragg, x. 72; Fellowes, p. 52; Louis, p. 194; Audouard, p. 56; Caisergues, p. 166; Amiel, p. 263; Boyd, p. 299; Blin, p. 6; Velasquez, p. 9; Pariset, Obs., p. 29; Gillkrest, ii. 270; Palloni, p. 4; Arejula, p. 159; Pariset, pp. 378, 384; O'Halloran, p. 82; Berthe, p. 82; Pym, p. 259; R. Jackson (Spain), p. 71; Imray, liii. 79; Pugnet, p. 353; Chisholm, i. 161; Ralph, ii. 65; McArthur, p. 346; Savarésy, p. 370; Osgood, p. 9; R. Jackson, i. 66, 104; Gillespie, p. 39; Barrington, xii. 311; Vatable, p. 345; Peixotto, i. 412; Catel, p. 11; Dariste, p. 160; Boyle, p. 203; Diet. des Sei. Méd., xv. 334; Wistar, pp. 9–11; Rochoux, pp. 299, 484; Maher, p. 842; Comrie, xiii. 169; Steward, iii. 187; Rufz, p. 13; Belcher, xxiii. 249; Musgrave, ix. 117; Arnold, p. 8; Evans, p. 256; Barry, p. 271; Levacher, p. 71; Jolivet, p. 9; Bourdon, p. 9; Joubert, p. 966; Cayenne (1850), pp. 166, 7; Anderson, p. 4; Carter, p. 3; Copland, iii. 139.

The pain affects at times, though not so frequently, the upper extremities, attacking sometimes one arm, sometimes both.¹

This pain, like the preceding, is felt during the stage of reaction, and subsides at the remission, returning sometimes—though not frequently—in the after stages of the disease (*Desportes*, i. 200). When acute, it is usually associated with the various grades of the inflammatory variety. It occasionally occurs, that after the close of an attack, the limbs become affected with acute pain. Speaking of the yellow fever of 1827 at Charleston, Dr. Dickson states, that “a few cases—all females—suffered during recovery the most severe pains in the limbs, both muscles and joints, like rheumatism, so violent as to cause them to scream and cry out, and to require for their relief very free and large doses of opiates.”²

The sensation of weariness, above alluded to, when connected with violent pain in the head, often changes to one of numbness, or tingling of the flesh. This is well calculated to excite fears relative to the safety of the patient; for it is almost always, if not invariably, the precursor of early dissolution (*Wragg*, p. 72).

4. *Stomach and Bowels*.—The gastric and abdominal organs are the seat of severe suffering in the disease before us. The pain therein experienced has always been regarded as forming an important link in the chain of morbid phenomena, constituting the disease, as it has manifested itself at various times in this city;³ while it has been equally noted in every other place where the fever has prevailed in an epidemic or sporadic form.⁴ The gastric pain

¹ Rochoux, pp. 299, 300; Drysdale, i. 128; Rush, iii. 66; Dariste, p. 160; Archer, v. 66; Caldwell, p. 81; Stone, vi. 556; Pariset, pp. 378, 384.

² Am. Journ., ii. 72.

³ Nassy, p. 21; Ffirth, p. 26; Rush, iii. 60–6, 206; Deveze, pp. 23, 24; Currie, pp. 24, 218; Barnwell, p. 370; S. Jackson, p. 55; Caldwell, p. 81; Pascalis, p. 37.

⁴ Drysdale, i. 134, 5; Dalmas, p. 6; Girardin, pp. 33, 55; Dickson, iii. 253, 4; Barton, pp. 9, 10; Brown, p. 14; Ticknor, iii. 222, 407; Townsend, pp. 147, 154, 161; Archer, v. 66; Irvine, p. 31; Tully, p. 295; A. Smith, xxv. 502; E. H. Smith, p. 118; Shecut, p. 119; Chabert, pp. 9, 10; Davidge, p. 103; Waring, p. 45; Drysdale, i. 134; Valentin, pp. 164, 5, 6; Baxter, xxi. 3, 4; C. Drake, xxi. 134; Revere, iii. 225; Merrill, ii. 222; A. Hosaek, p. 12; N. O. 1833, p. 334; Kelly, xiv. 377, 8, 9; Brown, p. 14; Harrison, ii. 135; Gros, p. 10; J. Warren, p. 501; Fellowes, pp. 53, 4; Louis, pp. 212, 229, 233; Audouard, pp. 57, 61, 63; Gillkrest, ii. 271; Proudfoot, xxvii. 249, 251; Caisergues, p. 166; Berthe, p. 82; Amiel, p. 264; Velasquez, p. 11; Pariset, Obs., p. 29; Palloni, p. 4; Dufour, iv. 50; Pariset, pp. 376, 7, 388, 399, 404; Pym, p. 229; R. Jackson (Spain), pp. 71, 79, 95; Arejula, p. 159; Thomas, p. 83; Stone, vi. 556; O'Halloran, pp. 79, 82, 126; Copland, iii. 154; Chisholm, i. 149; McArthur, pp. 346, 7; Bancroft, pp. 10, 124; Ralph, ii. 65, 67, 70; Savarésy, pp. 269, 273; Osgood, pp. 10, 13; Moseley, pp. 436, 8; Madrid, pp. 21, 25, 30; Blanc, p. 438; Gillespie, ii. 38; R. Jackson, Tr., p. 274; Hunter, p. 67; Pugnet, pp. 354, 5; Diet. des Sci. Méd., xv. 335, 7; Bally, pp. 217, 219; R. Jackson, Sketch, i. 68, 72, 93; Maher, p. 841; Arnold, p. 9; Dubreuil, viii. 321; Frost, pp. 30, 32; Comrie, p. 170; J. Clark, p. 8; Rochoux, pp. 286, 480; Caillot, pp. 18, 173; Evans, pp. 233, 256; Dyott, p. 1003; Belcher, xxiii. 249; Vatable, pp. 345, 352; Wilson, p. 8; Lempriere, ii. 64; Dickinson, pp. 127, 148; Grant, p. 31; Williams, p. 15; Hillary, p. 147; Dariste, pp. 159, 162; Desportes, i. 193, 199; Bruec, p. 279; Musgrave, ix. 134; Vincent, p. 24; Evans, p. 256; Blanc, p. 438; Lefoulon, p. 68; Finlay, p. 14; Cayenne (1850), p. 172; Lallemand, p. 88.

sometimes shows itself early, and attends on, and is aggravated by, the act of vomiting. Usually, however, it amounts, at this period, to a mere sensation of anxiety, uneasiness, heat, constriction or oppression at the epigastrium; and the more severe suffering comes on later than is the case with respect to the pain of the head, loins, or extremities—seldom before the accession of the third stage. More commonly, while the pain in the parts last mentioned, after being severe, during the first two or three days of the attack, subsides, more or less completely at the remission, seldom if ever to return in an intense form, the pain in the stomach, which had caused but little discomfort at first, comes on after the cessation of the period of calm, and continues to near the close of life, or the advent of convalescence. It must be remarked, also, that where gastric pain has existed in the early stage, it subsides with the other symptoms at the remission, but returns usually on the accession of the third stage.

At this period, the pain is of a burning, acrid, or griping spasmodic kind, and is accompanied with a feeling of weight and distension at the præcordia. Extending along the œsophagus to the pharynx, it is aggravated by motion or by the act of vomiting, and, in most cases, is a source of intolerable suffering—producing, in some instances, the most distressing state of body and mind. Dr. Rush states that, in 1793, it caused cries and shrieks, which were often heard on the opposite side of the streets to where the patients lay (iii. 66); and there is scarcely any one conversant with the disease, who has not witnessed instances of the kind.¹

“The stomach,” says Dr. Currie, “was constantly affected with a violent cardialgia, or a burning heat, which the miserable sufferers said felt as if scalded or burnt by a coal of fire; no posture gave ease, and whatever was swallowed, except very small draughts of toast and water, weak barley-water, or simple spring water, increased the agony to the most exquisite torture.”²

In this condition, the unhappy sufferer lies on his back or side, with his knees drawn up, obstinately refusing to change his position, or he stretches flat on his abdomen, or compresses it with his pillow or his hands.

The gastric pain just described is almost always accompanied with great tenderness of the epigastrium. This symptom, which has generally proved a prominent feature in the yellow fever, and has hence been pointed out by most writers,³ and considered as pathognomonic of the disease, contributes

¹ Audouard, Pariset, Rochoux, Jourdain, Vincent, Anderson.

² Fever of 1793, p. 30.

³ Rush, iii. 60, 206; Deveze, p. 24; Townsend, p. 161; Caldwell (1805), p. 81; Dariste, p. 159; Dickson, pp. 126, 7; T. Smith, xxxv. 42; R. Jackson (Spain), pp. 72, 81; Bertaud, p. 10; Thomas, p. 83; Gros, p. 10; Harrison, ii. 135; Louis, p. 212; Valentin, p. 164; Chabert, p. 8; Dariste, pp. 159, 162; O'Halloran, pp. 79, 82, 126; Frost, xiii. 32, 35; Comrie, xiii. 169, 170, 176; Gillkrest, ii. 270; A. Smith, xxv. 502; Rochoux, p. 287; McArthur, p. 346; Ralph, ii. p. 70; Wilson, p. 8; R. Jackson, i. 68, 72, 110; Thomas, p. 39; Bally, p. 217; Pariset, pp. 388, 399; Kelly, xiv. 377, 379; Dickinson, p. 127; Levacher, p. 72; Evans, pp. 232, 3; Girardin, p. 331; Williams, p. 16; Gillespie, p. 38; Townsend, pp. 154, 161; Barrington, xii. 312; Proudfoot, xxvii. 249; Blair, p. 64; Jolivet, p. 8; Bourdon, p. 9; Barnwell, p. 370; Berthe, p. 82; Caisergues, p. 166; Tully, p. 295; Copland, iii. 150.

to enhance the sufferings of the patient. It is sometimes excited by the slightest pressure of the part, by simply passing the finger over the skin, or by the weight of light fomentations, or even of the bedclothes. The least attempt to touch the part is resisted by the patient with all the expressions of intense agony and horror. But though very generally observed, it is not an inevitable attendant on the disease. In some epidemics, it prevails to a comparatively limited extent, or fails almost entirely, and in every epidemic cases occur, principally of the congestive kind, in which it is but slight or completely absent.¹

Much as has been said relative to the cause of this phenomenon, opinion is not as yet unanimous on the subject. That it is sometimes the effect of gastric inflammation we may infer from the fact that such inflammation exists, in many cases at least, and that this morbid state usually gives rise to tenderness of the epigastrium. But there is good reason to suppose that it is not always due to that cause. 1. It has been known to be intense in cases where, after death, very trifling or no lesions, resulting from previous inflammation, have been discovered. 2. In some cases, in which evidences of inflammation were incontrovertible, and were discovered after death, the tenderness in question failed to present itself, or existed in a very limited degree. 3. It is not experienced, or is so very feebly, in other acute diseases in which the mucous membrane of the stomach is inflamed to a greater extent. 4. The pain does not resemble that experienced in ordinary gastro-enteritic inflammation, but has been compared to that produced by pressure in peritonitis, a form of disease which does not exist in the yellow fever. With more propriety, it may be ascribed to a neuralgic condition of the parts, which, as we have seen, extends in some cases to the whole cutaneous surface.²

Dr. Rush remarks that, in 1793, very few complained, in the beginning of the disease, of that soreness to the touch about the pit of the stomach, which is taken notice of by authors, and which was universal in the yellow fever of 1762 (iii. 57). In 1820, it was not a universal attendant on the disease. "It is singular," says Dr. Jackson, "that the cases which occurred near Hodge's wharf should have been all nearly free from it. It was generally very slight with them. Pressure, made with force on the epigastrium, was borne without difficulty, though the stomach exhibited, by other symptoms, its highly disordered condition" (p. 55).

Occurring, in most cases, only after the accession of the third stage—between the third and the fifth day—tenderness at the epigastrium is necessarily absent in mild cases in which the disease ends before reaching that stage, and is often but slightly felt in those that recover after the supervention of the severer or even malignant symptoms. But it would be wrong to argue from this that epigastric tenderness is a sure indication of the benignity of

¹ Gilbert, p. 79; Pariset, p. 377; Harrison, ii. 133; Evans, p. 232; Gillkrest, p. 270; Louis, p. 214; Ashbel Smith, p. 501; Wragg, p. 72.

² Louis, pp. 213, 14; Harrison, p. 135.

the attack, and of the absence of much gastric derangement, as Dr. Rochoux would make us believe; for, independently of the fact that, in cases accompanied with cerebral engorgement, little or no pain appears to be felt in that part—whatever be the issue of the disease—it is not unfrequent to find the latter end fatally in cases in which pressure can be applied without an indication of pain being produced by it. Nor must we, on the other hand, regard it, except when it is excessive, as indicative of a fatal issue—inasmuch as cases end favourably which were attended with considerable epigastric tenderness.

Coinciding often, but not necessarily, with pain in the stomach, there is not unfrequently much pain about the umbilical region. Like the preceding, it makes its appearance at the close of the period of remission, towards the third or fourth day, and often causes much suffering. It is deep-seated—sometimes of a dull, but highly uncomfortable character—often it is acute and torturing. Together with this, patients often complain of a sense of burning pain and rawness in the pharynx and along the œsophagus, which, to many, is a source of great distress.

Connected with, and in great measure, occasioned by gastric and abdominal pain, we find the patient labouring often under an intolerable feeling of *malaise*, anxiety, distress, and anguish. This feeling, which he frequently refers to the epigastric region, is often associated with, and generally increased by nausea—but exists as frequently without it.¹ In many cases, it is attended with, and probably gives rise to those involuntary shrieks and cries, which have been alluded to in the preceding pages.

CHAPTER XV.

COUNTENANCE—COLORATION OF THE FACE AND EYES—URINE—RESTLESSNESS
AND JACTITATION—WAKEFULNESS—MUSCULAR POWER.

α. COUNTENANCE.—The discoloration of the face and eyes, to which attention has been called, associated, as it very usually is, with other changes in the condition of the parts, is combined with, or in great measure contributes in producing, a peculiar expression of countenance, which affords material

¹ Rush, iii. 66; Currie, p. 25; Gilbert, p. 65; Rufz, p. 14; Bayley, p. 93; McArthur, p. 346; Vatable, pp. 346, 352; Osgood, pp. 10, 13; Harrison, ii. 132; Pym, 229; Pariset, p. 392; Tully, p. 295; Berthe, p. 81; R. Jackson, i. 68, 69, 72; Ib., ii. 171, Tr., 254, 261, 274; Girardin, p. 53; Blanc, p. 438; Barton, pp. 9, 10; Desportes, i. 199; Copland, p. 155; Dancer, p. 83; Davidson, viii. 248; N. O. 1820, p. 9; Shecut, p. 119; Imray, liii. 81; E. H. Smith, p. 118; Monson, p. 180; Dickson, p. 256; Bally, p. 241; Hillary, p. 152; Gillespie, p. 41; Comrie, xiii. 170; Jolivet, p. 8; Jackson, Fev. of Spain, pp. 72, 84; Lefoulon, pp. 371, 2; Leblond, p. 103; Joubert, p. 966; Hume, p. 198; Copland, iii. 139, 140; Carter, p. 3.

aid towards the formation of a correct diagnosis and prognosis. In some cases, it is true, the countenance, though flushed, is otherwise unchanged in the commencement of the attack; and, occasionally, the expression remains unaltered to the last. But these instances, for causes assigned, are comparatively few in number; more commonly, the countenance is more or less altered. In many cases, of the inflammatory variety particularly, the face, during the stage of reaction, besides being red, is more or less swollen. This is observed principally about the nose and eyes, sometimes about the region of the parotid glands, and on the neck. The skin is tense, shining, and unctuous from excessive secretion of sebaceous matter.¹ This condition of the face varies in degree according to the violence of the attack, and generally to the constitution of the individual, and continues without abatement until the period of remission. It then, whatever may be the issue of the case, diminishes, and soon disappears completely—giving way, in the milder cases, or those that end favourably, to a healthful condition of the parts. In others of a more dangerous character, or fatal tendency, it leads to phenomena of a very different character. In the last stage of the disease, when the case has been at first inflammatory, and much earlier in the congestive variety—generally when the black vomit has been discharged in large quantities—the features become often shrunk, pinched, emaciated, elongated, and decomposed;² while, in some others, they assume a bloated, flabby, and inanimate appearance.³ From these various conditions of the features, combined, in many cases, with a contraction or knitting of the eyebrows—or, in others, with a drooping or half closed state of the lids—there result different and opposite expressions of countenance. In some instances of the first kind, we have the wild, ferocious aspect, which, as we have seen, Dr. Rush compared to that of a wild animal.⁴ As the disease advances, and the features become emaciated, the skin assumes a sombre, leaden, greenish-yellow, or livid hue, with red, sunken eyes, and ecchymosed lids; and the countenance presents a ghastly aspect, with haggard eyes, frightful to behold. “The face of a lady,” says Dr. Rush, “admired when in health for uncommon beauty, was so much distorted by the commotion of her whole system as to be viewed with horror by all her friends.”⁵

In cases presenting this ghastly and haggard appearance, and in other

¹ Rochoux, p. 294; Copland, iii. 156; Drysdale, i. 29; Blane, p. 435; Savarésy, p. 289; R. Jackson (Spain), p. 95; Ticknor, iii. 224; Merrill, ix. 244; Dickson, i. 354; A. Smith, xxv. 501; Louis, pp. 167–8; Arejula, Ed. J., i. 449.

² Savarésy, p. 277; Gillkrest, ii. 271; Ruz, p. 15; Comrie, xiii. 176; Dict. des Sci. Méd., xv. 335; Furlong, p. 29; Belcher, xxiii. 335; Rochoux, p. 558; R. Jackson, i. 84, 184; Ib., Fev. of Spain, p. 62; Copland, iii. 156.

³ Jackson, i. 108; Townsend, p. 165.

⁴ Rush, iii. 52; Hill, v. 87; Davidge, p. 103; Comrie, xiii. 176; Davidson, i. 249; Rochoux, i. 149; Hunter, p. 69; Osgood, pp. 9, 10; Jourdain, v. 258; R. Jackson (Spain), pp. 73, 80; Townsend, pp. 147, 155; Chisholm, i. 149.

⁵ Dalmas, p. 6; Davidson, i. 249; R. Jackson, i. 94; Gros, p. 12; Lempriere, ii. 845; Rochoux, p. 489; Bally, pp. 214, 235; Audouard, pp. 61–2; Gillkrest, ii. 271; Dariste, p. 165.

instances of a different kind, the countenance is characterized by an expression of suffering, dejection, anxiety, anguish, despair, or terror.¹ "The countenance is peculiar, and not easily definable by words, having an inanimate sadness of expression, which, with the state of the eye, constitutes diagnostic marks of a disease difficult to be mistaken by those who have once seen it."² In others, again—generally later—it is marked by an air of stupidity, vacaney, astonishment, sullenness, dulness, and indifference;³ while, in the latter stage, it is not uncommon to find the countenance as expressive of entire resignation, as it had been before of anxiety and terror. The following is a faithful picture of what was seen in this city during the epidemic of 1820, and of what will be found to apply to the disease at other times and in other places: "The physiognomy," says Dr. Jackson, "had something peculiar and striking. It conveyed at once an impression of the malignant nature of the disease, and the danger of the patient. It is impossible to describe the appearance of the eyes and features; the look and expression that seemed to indicate an internal consciousness of the mortal struggle in which the vital powers were engaged; or the scowl of a gloomy indifference that mantled the brow, and shrouded the countenance with the expression of a sullen defiance of the fate that seemed impending over them with certain destruction; but once seen, it could not be forgotten."

On this subject, Dr. Lewis, of Mobile, who is entitled to our regard in all that relates to the yellow fever, remarks that the physiognomy of the diseases which he has seen, with the exception of yellow fever, is usually associated in his mind with the peculiar symptoms of the malady, or rather would seem to be the mere shadowing of the strong and well marked phenomena that distinguish them—such, for instance, as is witnessed in congestive fever, cholera, and tetanus. But the physiognomy of the yellow fever cannot be referred to symptoms. It would appear *a fiendish something* independent of, and beyond the ordinary phenomena of the disease. In many cases it is stamped upon the brow at an early period. Once enthroned, no effort of the patient can disturb its reign; he may smile and laugh, but cannot chase it away. There it still sits, paralyzing those reacting efforts of nature which are ever struggling against disease, and mocking the assumed gayety and levity of its victim. Even the cradle is not exempt from its visitations; while, within the last hour, I have seen a child but fifteen months old, over whose brow this mysterious fiend had spread his gloomy mantle, giving to

¹ Rush, iii. 52; Caldwell, p. 83; N. O. 1820, p. 8; Barton, p. 10; Dickinson, pp. 132-4; Dict. des Sci. Méd., xv. 335; Ruz, p. 15; N. O. 1839, p. 332; Vatable, p. 345; Barrington, xii. 311; Blane, p. 436; Lempriere, ii. 60, 70, 84; Osgood, pp. 9, 10; McArthur, p. 348; Ralph, ii. 66-7; Dariste, p. 159; Peixotto, i. 412; Bally, pp. 214, 225; Davidson, i. 249; Dancer, p. 82; R. Jackson, i. 69-111; Wilson, pp. 11, 13; Drysdale, i. 29; Gros, p. 9; Pariset, p. 387; Townsend, p. 155; Hayne, vii. 8.

² O'Halloran, Fev. of Andalusia, p. 81.

³ Jackson, i. 93, 105; Rochoux, p. 489; Caldwell, p. 83; Pariset, p. 397; Gillkrest, ii. 271; Dalmas, p. 6; Kelly, xiv. 379; Ralph, ii. 72; Merrill, ii. 245.

the little patient a dejected, cheerless, and earnest look, illy suited to its infant face."¹

Finally, the closing stages of the disease are not unfrequently marked by tremor, agitation, twitching, and even convulsive movements in the muscles of the face and lips.²

b. COLORATION OF THE FACE.—In cases in which the attack is not sudden, and is preceded by signs of approaching disease, the face at this period is often found more or less altered in appearance—pale, dusky, or red, and affected sometimes with transient flushes. During the cold period or the chill, when the latter occurs, the face is, as in other complaints of kindred character, more or less pale, and sometimes livid. But, whatever may have been its appearance previous to the attack, the face, a few hours after the latter has fairly set in, becomes generally highly flushed, of a deep red and preternaturally florid colour. The suffusion is spread all over, though more particularly on the cheeks, and sometimes assumes a crimson or violet like dusky hue; and at others is so intense as to produce the appearance of inflammation.

This red coloration of the face, which in the yellow fever far exceeds the degree it attains in other febrile affections, has been noted, to a greater or less extent, in all the epidemics with which this city has been visited, as will be found recorded in the writings of Rush (iii. 56, 212), Currie (p. 21), Cathrall (p. 24), Nassy (p. 20), Deveze (p. 22), Jackson (pp. 51, 59, 61), and Caldwell (1805, p. 83).

It is found to manifest itself in the fever of other cities of this country,³ has been observed in Europe,⁴ and is characteristic of the disease in tropical regions.⁵ In this respect, indeed, our fever approximates closely not only to the yellow fever of Spain and Italy, but also to that of the West Indies and

¹ N. O. J., i. 299.

² Imray, liii. 81; Lempriere, ii. 66; Gillkrest, ii. 271; Bancroft, p. 13; Rochoux, p. 558; Audouard, p. 64; Chisholm, i. 163; Jolivet, p. 9.

³ Dalmas, p. 6; Townsend, p. 147; Thomas, p. 82; Girardin, 33; Perlee, iii. 11; Dickson, Essay, i. 348; *Ib.*, Med. and Phys. Journ., iii. 254; Archer, v. 66; A. Smith, xxv. 501; Drysdale, i. 24; Lining, ii. 414; Simons, p. 7; Nott, Charleston Journ., iii. 9; Ticknor, iii. 224; Hill, v. 89; Gros, p. 7; New Orleans in 1819, p. 8; E. H. Smith, p. 115; Monson, p. 179; New Orleans in 1820, p. 8; *Ib.*, in 1839, p. 331; Kelly, xiv. 377; Moultrie, p. 3; Wragg, x. 72; Randolph, Med. Repos., xxiii. 167.

⁴ Pariset, p. 384; Arejula, Edinb. Journ., i. 448; Velasquez, p. 10; Louis, pp. 167, 168; Pariset, Obs., p. 27; Audouard, pp. 56, 57; Caisergues, 167; Pym, pp. 228, 229, 233; Berthe, p. 81; Copland, iii. 141.

⁵ Bruce, 278; Hunter, p. 65; Fontana, p. 72; Pugnet, 354; Vatable, p. 345; Rufz, p. 54; Gillespie, p. 38; Hillary, p. 147; Bancroft, p. 30; Moseley, p. 436; Desportes, i. 194; Imray, liii. 79; Osgood, p. 9; McArthur, p. 346; Chisholm, i. 149; Savarésy, p. 269; Frost, xiii. 29; Ralph, ii. 70; Levacher, p. 71; Rochoux, pp. 294, 295; R. Jackson, i. 65, 66; Dickson, in Johnson, p. 365; Dancer, p. 82; Bally, p. 214; Wilson, p. 8; Maher, pp. 842–848; Gilbert, p. 65; Catel, p. 11; Davidson, Repos., viii. 248; Dariste, pp. 158, 159; Blair, p. 64; Evans, p. 256; Leblond, p. 102; Vincent, p. 23; Mabit, p. 10; Jolivet, p. 9; Dupont, p. 18; Bourdon, p. 9; Joubert, p. 966; Carter, p. 4; Cayenne, (1850), p. 174; Catel, p. 224; Bone, p. 5; Simons, p. 7; Copland, p. 141; Randolph, Med. Rep., xxiii. 167; Anderson, p. 5.

of the African coast; for, in the description we have of these, the writers employ often the same terms to denote the appearance of the face that are made use of in this country and in other parts of temperate regions. There, as with us, it is highly suffused, of a deep red, crimson, purplish, or violet colour; there, as with us, it imparts the idea of inflammation.

This condition of the face continues, with little or no change, during the whole of the period of reaction; and, beginning to subside on the appearance of the remission—on the third or fourth day, sometimes earlier, rarely later—soon disappears, and gives place to the jaundiced discoloration to which attention has already been called. In some cases, the cheeks remain red while the rest of the face becomes yellow, and in many there is a mixture of red and yellow. To this change from a red to a yellow hue, which contributes towards marking the accession of the second stage, I have already referred in a former chapter, and need not recur to it here.

Connected as this appearance of the face is with the state of vascular reaction or excitement denoting the existence of the first stage, and aware, as we must be, that a greater or less degree of such a reaction manifests itself in a large number, if not a majority, of cases, we may readily infer that the symptom in question is one of general occurrence. But, although such is the fact in every place where the yellow fever prevails, and although, consequently, the phenomenon may be considered as characteristic of the disease, yet nevertheless cases present themselves, more frequently in some epidemics than in others, in which the face becomes but slightly red, or presents a rosy or Circassian hue; others, in which it remains of the natural colour, or becomes paler than in a state of health; and others, again, in which it assumes a livid or clay, ash, dingy, lurid, or putrid sallow hue. In some cases, this peculiar appearance of the face continues to the close of the attack; while in others it is replaced by, or combined with, a yellow or mahogany tinge of the skin.¹

It need scarcely be added that the peculiar red suffusion of the face appertains to the several grades of the inflammatory form of the disease, and the pale, livid hue just noticed to the congestive and adynamic forms. The latter is indicative of more danger than the former; while the degree to which both are carried is a pretty sure criterion of the severity of the case.

c. COLORATION OF THE EYES.—Characteristic as may be the particular appearance of the face in the early stage of the yellow fever, it is less so than that of the eyes, changes in which have attracted the attention of medical and other inquirers from the days of Du Tertre and Labat to our own. Whatever may be the appearance of those organs during the premonitory or cold stage of the disease, reaction, however slight, has scarcely set in, before they become remarkably red, or even bloodshot, hot, more or less intolerant of light, and otherwise painful, imparting a sensation similar to that produced by the introduction of grit or sand. In many instances, the balls of the eyes appear to consist of a mass of vessels gorged with blood; they are, at the same time,

¹ Pym, p. 230; Bally, p. 214; Wilson, p. 11; R. Jackson, i. 87, 91, 104, 105, 108; Madrid, p. 6; O'Halloran, p. 124; Kelly, p. 378; Rochoux, p. 467; Copland, iii. 144.

brilliant, shining, and watery. Presenting sometimes the expression peculiar to drunkenness, they in many other cases assume an inflamed, fiery appearance, which has been not unaptly compared to that of a wildcat, or a cat in the dark. In others, again, from the half-closed lids, they present an aspect of distress. This peculiar appearance of the eyes, as has often been remarked, is easily recognized, but with difficulty described; and, when found, as it often is, associated with entire integrity of the intellectual functions, is well calculated to prove a subject of interest to the pathologist. It has been noticed, to a greater or less extent, in all our epidemics, and is graphically described in the accounts we have of these.¹ These descriptions differ but slightly, if at all, from those we have of the morbid aspect and peculiar expression of the same organs in the yellow fever of other parts of the United States, from Boston to Galveston.² As might be expected, the aspect of the eyes in the yellow fever of Europe bears a strong analogy to what has been observed in this country. Tommasini (i. 9), Palloni (p. 4), Dufour (iv. 50), Moechi, Pasquetti, and Brignole³ unite in stating that these organs, in the first stage of the disease, as it appeared at Leghorn in 1804, were fiery, flashy, highly injected, and, as it were, inflamed. Like phenomena are recorded by those who have observed the yellow fever at Gibraltar and the various cities of Spain;⁴ and those who are conversant with the writings of tropical physicians, need scarcely be told that in those regions, whether in the West Indies or on the coast of Africa, the eyes present the same aspect;⁵ a circumstance which, as much as anything else

¹ Rush, iii. 52-56; Currie, p. 21; Fever of 1797, iv. 42, 129; Cathrall, p. 22; Barnwell, p. 369; Caldwell (1805), p. 83; Monges, p. 56; Pascalis, p. 30; Deveze, p. 22; Jackson, p. 52; Firth, p. 26.

² Girardin, pp. 33-55; Drysdale, i. 29, 133; Valentin, p. 166; Barton, pp. 9, 10; Lining, ii. 414-421; Baxter, xxi. 3; Townsend, pp. 144, 172; Dalmas, p. 7; Cartwright, ix. 12; Kelly, xiv. 377; Perlee, iii. 11; Moultrie, p. 3; Ticknor, iii. 224; Monson, p. 179; Hill, v. 89; Seaman, p. 10, iv. 249; Archer, v. 66; E. H. Smith, p. 115; Tully, p. 295; Gros, p. 9; New Orleans in 1819, p. 8; Ib. in 1839, p. 331; Ib. in 1820, p. 8; A. Hosack, p. 11; A. Smith, xxv. 501; Shecut, p. 119; Stone, vi. 559; Bayley, pp. 92-96; Merrill, ix. 244; Dickson, iii. 253, Essays, i. 348; Barrington, xii. 311; Davidge, p. 102; Simons, p. 7; Wragg, x. 72.

³ *Relazione medica della malattia che domina presentement in Livorno.*

⁴ Pym, pp. 228, 229; Amiel, in Johnson, p. 263; Louis, pp. 168, 172; Arejula, Edinb. Journ., i. 448; Caisergues, p. 166; Gilpin, v. 322; Velasquez, p. 10; Smith, xxxv. 42; Pariset, Obs., p. 29; Gillkrest, pp. 270-272; Berthe, p. 81; Fellowes, pp. 53, 280; Proudfoot, xxvii. 252; R. Jackson, 60, 61, 97; Audouard, pp. 56-58; O'Halloran, pp. 77, 78; Pariset, &c., p. 385; Jourdain, v. 259; De Maria, p. 65.

⁵ Bruce, p. 278; Chisholm, i. 149; Pugnet, p. 354; Hunter, p. 66; Vatable, p. 345; Jackson, Tr., p. 254; Ib., Sketch, i. 65; Gillespie, p. 38; Hillary, p. 147; Bancroft, p. 9; Moseley, p. 436; Desportes, i. 194; Imray, liii. 79; McArthur, p. 346; Osgood, p. 9; Ralph, ii. 68; Savarésy, p. 269; Peixotto, i. 412; Frost, xiii. 29; Comrie, xiii. 175; Levacher, p. 71; Wilson, p. 8; Caillot, p. 16; Dickinson, p. 125; Musgrave, ix. 117; Rufz, p. 11; Maher, pp. 842-848; Gilbert, p. 65; Dancer, p. 82; J. Clark, pp. 6-8; Catel, p. 11; Morgan, iv. 4; Dariste, p. 158; Bally, pp. 213, 214; Dict. des Sci. Méd., xv. 334; Dyott, p. 1003; Belcher, p. 250; Warren, p. 9; Dickson, in Johnson, p. 365; Barry, in Boyle, p. 271; Rochoux, p. 295; Doughty, p. 11; Madrid, pt. ii. p. 24; Copland, iii. 156; Leffoulon, pp. 67, 68; Leblond, pp. 102, 103; Mabit, p. 10; Jolivet, p. 9; Dupont, p. 18;

proves the identity of that fever with that of temperate regions. There, as with us, we find that aspect characterized as produced by suffusion, turgidity of the bloodvessels, and as imparting the idea of inflammation; there, as with us, the eyes are said to be brilliant, sparkling, humid, swollen, and prominent; there, as with us, too, these appearances of the eyes are associated with pain in the balls, burning sensation of the lids, and an intolerance of light; and while Dr. R. Jackson states that in the West Indies the eye often appears as if it had been exposed to the smoke of gumwood,¹ he uses the same comparison in relation to the fever of Spain, which he had occasion to witness in 1820 (pp. 60, 61); and a physician of our country—Dr. Dickson, of Charleston—thinks it applies well to the state of the organ as observed in that city in 1817.²

This peculiar appearance of the eyes, which, when added to that of the face, extorted from Dr. Rush the remark that “it was as much unlike that which is exhibited in the common bilious fever as the face of a wild is unlike the face of a mild domestic animal” (iii. 52), may be regarded as characteristic of the yellow fever, and alone often enables the physician to distinguish, at first glance, that disease from other forms of febrile complaints. Like the red appearance of the face, it increases until about the third day, and is generally proportioned to the severity of the attack. It subsides about the period of the remission or metaptosis, when it gives way to a yellow or orange tinge, which goes on deepening until it assumes a decided jaundice hue—or it forms a compound with the latter; while the eyes, losing their glistening appearance, gradually acquire, in fatal cases, an expression of languor and distress, sink in their sockets, and are surrounded by a livid circle. Like the redness of the face, also, that of the eyes, although existing in the greater number of cases, fails partially or completely in others, when these organs assume a dull reddish, or yellowish red, glassy, or drunken, vacant, idiotic appearance. In others, the colour is of a dingy or muddy character. In others, again, the eyes retain their natural appearance, but are difficult to move. In another set, again, they are white, even pearl white, or of a white dusky yellow, and withal glossy and painful. In some, also, the conjunctiva presents a bluish cast, and is surcharged with blue vessels, while the cornea is watery and glistening.³

The deep red or inflamed eye above described appertains—though not exclusively—to the inflammatory form of the disease, instances being occasionally encountered in which it accompanies, though in a less degree, especially so far as concerns the brilliancy and glistening appearance of the

Bourdon, p. 9; Cayenne (1820), p. 175; Catel, p. 224; Bone, p. 4; Finlay, p. 14; Simons, p. 7; Hume, p. 197; Grant, p. 29; Heastie, p. 18; Copland, iii. 139; Carter, pp. 3, 4; Anderson, p. 5.

¹ Sketch, i. p. 65.

² Phila. Journ., iii. 253.

³ R. Jackson (Spain), p. 61; Kelly, xiv. 378; Barton, p. 9; Velasquez, p. 13; R. Jackson, Sketch, i. 87, 91, 105, 108; Merrill, ix. 245, ii. 223; Rochoux, p. 487; O'Halloran, p. 124.

organ, the congestive and adynamic forms; while the latter is usually attended by some of the morbid conditions last enumerated.

It remains to be mentioned that, in many cases—particularly of the congestive and adynamic kind—the pupils are found more or less dilated.¹ This dilatation often appears in the first stage of the disease—sometimes occurs before the attack—and even is noticed in some individuals who, though exposed to the epidemic cause, entirely escape. In other instances, it is not noticed before the second or third stage of the disease. But, at whatever period it may appear, it continues to the close of the case. Dr. Rush, who was one of the first to call attention to the state of the pupils, attributed their dilatation to some affection of the brain, which he called “the *phrenicula*, or inflammatory state of the internal dropsy of the brain.” This he suspected from the slow, corded, and intermittent pulse with which he found it associated, and from the known effects of affections of the brain in enlarging the pupil. Dr. Chisholm, also, who about the same time noticed the frequent occurrence of this dilatation, referred it to congestion of the brain, and thought it was always associated with coma. But, true as it undoubtedly is, that the dilatation of the pupil may and does depend sometimes on the cause thus assigned to it, we can find no difficulty in perceiving, from the facts mentioned, that the explanation is not applicable to all cases. Its occurrence in the first stage, in which the absence of all cerebral congestion is so common—its occurrence as a premonitory symptom when such a congestion is still less frequent, and its very general prevalence among individuals exposed to the action of the poison, but otherwise enjoying good health—a fact dwelt upon by Dr. Rush himself—all these circumstances forbid the admission of the exclusive agency of cerebral compression, and lead to the supposition of its being generally due to the sedative effect of the morbid poison giving rise to the disease.

d. URINE.—In the yellow fever, the urine, particularly during the early stages of the disease, does not assume, as it does in some other forms of febrile complaints, features of a pathognomonic character, varying, as it often has been found to do in point of appearance and quantity, not only in this city and country, but in Europe and tropical regions.

In many cases it presents, at the outset—and, indeed, throughout the first stage—its natural aspect; sometimes it is paler and more limpid than in health.² This natural state of the urinary secretion, which is found to continue during the whole of the first stage, loses often—as Lining long ago

¹ Townsend, p. 144; Monson, p. 179; Dict. des Sci. Méd., xv. 334; Rush, iii. 55; Gros, p. 12; Chisholm, i. 155–6; Kelly, xiv. 378; Imray, liii. 81; Rufz, pp. 11, 13; Gillespie, p. 43; Wilson, p. 11; Barton, p. 9; Drysdale, i. 126; Gilpin, v. 322; Catel, p. 11; Bally, pp. 213, 214; Dickinson, pp. 130, 135.

² Rush, ii. 62; Bayley, p. 78; Townsend, p. 147; Lining, ii. 415; Moultrie, p. 7; Desportes, i. 194; Rochoux, pp. 328, 509; Louis, pp. 235–240; N. O. 1839, p. 332; Pariset, pp. 373–423; Velasquez, pp. 10–13; Caillot, p. 18; Ralph, ii. 66; Lefoulon, pp. 69, 371; Flores, p. 51; Lallemand, p. 110.

particularly pointed out—its paleness and limpidness after the first day, and is discharged less freely—the change being independent of the quantity or quality of the liquids drunk. This peculiar condition of the urine is an attendant either on mild cases, in which the reaction does not run high; or, not unfrequently, on those of a congestive character, when the arterial system is not excited, or is even depressed, and the skin is cool. More frequently it is replaced, especially in the inflammatory form of the disease, by opposite conditions. In these cases the urine is found red, or high-coloured, and remains so till the accession of the second stage, the hue varying from a light to a deep red, and usually deepening in proportion to the severity of the attack. This high coloration of the urine, which, as is well known, is not peculiar to the yellow fever, but presents itself in other fevers of an inflammatory form, has been noticed, not in this city alone, but in all places visited by the disease, and has been very generally pointed out by our own and other writers. It has been observed in this city in all our epidemics.¹ In the fever of other parts of this country it has not been less conspicuous;² while in Europe,³ and in tropical regions,⁴ it has ever constituted a prominent symptom.

As the disease advances, a notable change usually takes place in the urine, which then presents peculiarities not generally observed in other fevers. At the accession of the second stage, though sometimes retaining the red hue just mentioned,⁵ it more frequently assumes a yellowish tinge, more or less marked,⁶ and often proportionate to the extent of the jaundice. This peculiar condition of the secretion is not only perceptible on simple inspection, but is made still more manifest by the effect produced on paper or muslin dipped in, or stained by it. It occurs in cases which end favourably, as well as in others of a different kind; and often continues unchanged for some days after recovery. This colour of the urine depends on the presence of bile or its constituent principles, as has been satisfactorily ascer-

¹ Rush, pp. 62–207; Currie, p. 21; Deveze, p. 23; S. Jackson, pp. 56, 60; Nassy, p. 22.

² Drysdale, i. 136; N. O. 1820, p. 8; Lining, ii. 415; A. Hosack, p. 12; Townsend, p. 147; Davidge, p. 103; Moultrie, p. 3; Harrison, ii. 131; N. O. 1839, p. 432; Stone, vi. 561; Gros, pp. 9, 10; Girardin, p. 33; Hill, v. 90; Ticknor, iii. 224.

³ R. Jackson (Spain), p. 73; Audouard, p. 57; Proudfoot, xxvii. 250; Amiel (Johnson), p. 263; Pariset, Obs., p. 29; Velasquez, p. 12; Rochoux, p. 509.

⁴ Lempriere, ii. 90; Ralph, ii. 65; McArthur, p. 346; Savarésy, p. 270; Moseley, p. 436; Rochoux, p. 328; Gillespie, p. 42; Davidson, viii. 248; R. Jackson, Sketch, i. 63, 67, 88, 89, 108, 173; Ib., Tr., p. 290; Peixotto, i. 413; Maher, p. 843; Dariste, p. 160; Rufz, p. 54; Belcher, p. 250; Warren, p. 9; Holliday, p. 9; Dickinson, p. 126; Chisholm, i. 173; Hillary, p. 152; Wilson, p. 9; Grant, p. 32; Desportes, i. 194; Le-foulon, p. 69, 371.

⁵ N. O. 1839, p. 333; Palloni, p. 4.

⁶ Moultrie, p. 4; Townsend, p. 155; C. Drake, xxi. 134; A. Hosack, p. 13; Lining, ii. 423; Hayne, vii. 7; Drysdale, i. 136; Stone, vi. 561; Dufour, iv. 51; Audouard, p. 58; Velasquez, p. 10; Rochoux, pp. 329, 509; Caillot, p. 18; Bally, p. 227; Hillary, p. 152; Evans, p. 245; Bruce, pp. 278–9; Hunter, p. 72; Finlay, p. 16; Copland, iii. 165.

tained through means of chemical tests, by myself and many others.¹ Dr. Wragg remarks that the quantity of bile found to be eliminated from the system in this way is immense (p. 78). In the last stage of the disease, the urine very generally presents a dark appearance, varying much, in different cases, in point of hue, being of a deep red in some, or of a green, brown, coffee, porter, or port-wine colour, or black in others; whilst, in many cases, it is evidently mixed with dark blood, pure or grumous, or with matter similar to that of the black vomit, which falls to the bottom of the vessel in the form of a dark grumous sediment.²

This dark colour of the urine is doubtless due, in most cases, to a greater or less admixture of blood with the secreted fluid. It shows itself at a period when passive hemorrhages manifest themselves, and has been found to be vicarious of the black vomit, when the kidneys and bladder partake extensively in the morbid changes of the disease.³ Though often the precursor of a fatal issue, it is found to occur in cases that terminate favourably, and sometimes continues several days after recovery.⁴ In many cases the urine presents, at this period of the disease, an offensive ammoniacal smell, and is found sometimes acid, with a strong action on litmus paper.⁵ This acid reaction would appear to depend on the presence of hydrochloric acid, the quantity of which carried off in this way is at times very considerable. Dr. Wragg, who has verified this fact in the cases treated by him in the Roper Hospital, Charleston, during the epidemic of 1854, is disposed to believe, from the frequent presence of this acid in the stomach and intestines during the early stage of the fever, that its presence in the urine takes place earlier, and is more frequent than we are yet aware of. "It has not been possible for me," says this able physician, "to institute a comparison between the number of deaths and recoveries in cases where this state of the urine was made out. It appears to have been coincident with great danger to the patient; but it will be a point of much interest to be investigated hereafter, whether its presence in the urine at this stage of the disease is not, as in the case of bile, under the same circumstances, proof of an eliminative action."⁶

The urine, though in some cases clear and limpid at the commencement, and even through the greatest part of the disease, is more frequently more or less cloudy, obscure, thick, and without sediment. At other times, it is

¹ Copland, iii. 165; Finlay, p. 16.

² Rush, iii. 62; Nassy, p. 24; Bayley, p. 98; Deveze, p. 26; Moultrie, p. 5; Stone, vi. 562; Pariset, p. 423; Velasquez, p. 12; Audouard, p. 58; Bruce, p. 279; Chisholm, i. 173; Moseley, p. 436; Lempriere, ii. 65; Hillary, p. 152; Blane, pp. 440; Bancroft, p. 13; Maher, p. 843; Levacher, p. 72; Dict. des Sci. Méd., xv. 337; Desportes, i. 194; Holliday, p. 9; Caillot, p. 10; Grant, p. 33; Blair, p. 66; Evans, p. 245; Pugnet, p. 360; Rochoux, pp. 328-509.

³ Blair, p. 66; Levacher, p. 73.

⁴ Rush, iii. 62.

⁵ Moultrie, p. 4; Chisholm, i. 173; Bancroft, p. 13; Holliday, p. 9; Caillot, p. 19; Levacher, p. 72; Dict. des Sci. Méd., pp. 15, 337.

⁶ Charleston J., x. 78-9.

sedimentitious.¹ It has been found to present, in the second stage, a greasy appearance on the surface, or to be covered with shining particles.²

Dr. Rush and other writers mention cases in which the patients have voided large quantities of urine a short time before death (iii. 62; iv. 41); and, in many instances, particularly when the fluid is of a natural or pale colour, the quantity discharged is, from the commencement and throughout, plentiful. More commonly, however, the secretion is, from the commencement, scanty, and becomes more so as the disease advances,³ and the discharge of it attended with a scalding pain and a difficulty amounting, at times, to dysuria.⁴

When the urine is scanty, it appears like turbid serum, with small particles apparently of lymph, floating through it. It has neither urinous nor aromatic odour. Dr. Pennell found it very coagulable. In some instances, he has known the urine, when heated, to become an entire solid mass.⁵ The author of the present work has made, several times, the same observation, and, indeed, is not disinclined to unite in opinion with those who believe that it rarely happens that a patient suffering from the yellow fever has not albuminous urine. In the slightest cases, the albumen, as Dr. Pennell remarks, may be, and generally is, so small in quantity as to be scarcely discernible; but, in every instance, where the fever is well-marked, the presence of albumen is readily detected. In fatal cases, this state of urine is accompanied by a condition of the kidneys not unlike that witnessed in Bright's disease. The condition of both secretion and organ accords with the state of the gastric enteritic mucous membrane, and the passive hemorrhages to which it is subject. In Barbadoes, also, the urine has been found to be albuminous. Dr. Collins, in a letter to Dr. J. Davy,⁶ says that this was discovered in every case of decided yellow fever. This condition was assumed about the second or third day, and was maintained throughout, increasing as the disease advanced. In cases of protracted convalescence, it continued long after all symptoms but debility had left the patient. Most of the albuminous precipitates, Dr. Davy adds, on the authority of Dr. Collins, were of a brownish colour; in some cases, just before the fatal termination, the presence of blood in the

¹ Currie, p. 21; Rush, p. 62; Deveze, p. 24; S. Jackson, pp. 56, 60; Hill, v. 90; Lining, ii. 415, 416; Bayley, p. 98; R. Jackson Tr., p. 259; Velasquez, p. 10; Rochoux, p. 328; Levacher, p. 72; Moultrie, p. 4; Drysdale, i. 136; Desportes, i. 194; Maher, p. 843; Caillot, p. 18.

² Bally, p. 227; Deveze, p. 22; Stone, vi. 562.

³ Rush, p. 207; S. Jackson, p. 56; Currie, p. 21; Deveze, p. 23; Davidge, p. 103; A. Hosack, pp. 12, 16; Irvine, p. 26; J. Warren, p. 502; N. O., 1820, p. 8; C. Drake, xxi. 134; Merrill, ii. 222; Bayley, p. 98; Townsend, p. 147; Palloni, p. 4; O'Halloran, p. 82; R. Jackson (Spain), p. 73; Proudfoot, xxvii. 250; Amiel (Johnson), p. 263; Moseley, p. 436; Ralph, ii. 66; Davidson, viii. 248; Peixotto, i. 413; Levacher, p. 72; Rufz, pp. 15, 54; Wilson, p. 9; Belcher, p. 250; Evans, p. 245; Caillot, p. 18; Holliday, p. 9; Blane, p. 430; Savarésy, p. 270; Grant, p. 33.

⁴ Rush, p. 62; Deveze, p. 23; Harrison, ii. 131; Stone, vi. 562; C. Drake, xxi. 135; O'Halloran, p. 82; Dickinson, p. 126; Savarésy, p. 270; Wilson, p. 9; Merrill, ii. 222.

⁵ Med.-Chir. Tr., xxxvi. 52.

⁶ See Blair, p. 99, note.

urine was recognized by the microscope. In two instances, Dr. C. found the albumen in the urine replaced by a substance having the properties of caseine.

On chemical analysis, urine taken from the bladder after death has been found greatly deficient in the proportion of urea. In one case, 200 grammes (51 drachms) of the fluid contained but one gramme and $\frac{90}{100}$ (about 16 grains) of urea; no uric acid, and 0.45 per cent. of albumen. In another case, 15 grammes ($\frac{1}{2}$ oz.) of the fluid obtained in the same way, gave 0.08 of urea, 0.50 of albumen, and no uric acid. In several other experiments, conducted in the same way, the results were similar. Finally, in one case, the urine was examined in the first stage of the disease, and a short time after the death of the patient. In the first, the fluid was found to contain, in 100 parts :—

Water	76.08
Urea	2.64
Albumen	0.40
Uric acid	0.08
Earthy phosphates, sulphates, alkaline phosphates, and chlorates	0.80
	<hr/> 80.00

After death, 20 grammes gave mere traces of urea; 0.50 of albumen, and no uric acid.¹

This shows a great diminution of urea, which exists in urine voided by an individual in health in the proportion of from 7.568 to over 30 in 1,000—the average being about 12, according to the amount of water drank, and consequently to the degree of concentration of the fluid—100 parts of the solid matter containing from 30.07 to 49.91 of urea.² The diminution is greater than in malarial fevers generally, in which the average is about 9. If the experiments I have referred to are reliable, the urine of yellow fever differs from that of other varieties of such fevers, in which the uric acid is in larger quantity than in the former.

In a few cases the urine, during the last stage of the disease, is passed off involuntarily.³ In others, it is retained and accumulates in the bladder,⁴ owing apparently to a paralytic condition of that organ; while, in a number of instances, its secretion is completely, or, in very great measure, suppressed. This peculiar morbid change, which must not be confounded with the preceding, is attributable sometimes to the inflammatory state, which, as we have seen, occurs in the kidneys, to the blocking up of the tubuli uriniferi by exudation-matter, or to a condition similar to that witnessed in Bright's disease; but, in other instances, perhaps the majority, it is evidently due to a simple suspension of the secretory function of those organs from nervous atony, and is not connected with any appreciable morbid state of the glandular tissue. Though not exclusively peculiar to the yellow fever, having

¹ Chassaniel, Ac. of Sc., Sitting of 12 Dec. 1853.

² Simons, ii. pp. 143-7.

³ Deveze, p. 26; Bancroft, p. 14.

⁴ Jackson, p. 56; R. Jackson.

been observed in the oriental plague, in malignant fevers and in some malignant cases of smallpox, it is more frequently an attendant on the closing stage of the yellow fever than of any other disease; and being an almost sure indication of approaching death, has been noticed by every writer on the disease, both among us and abroad.¹ Dr. Pennell tells us that out of many hundreds, whom he treated at Rio during the epidemic of 1850, he cannot recollect but one instance in which it was ascertained by the catheter that suppression of urine existed for twenty-four hours, and that the patient recovered.² Dr. Wragg was somewhat less unfortunate. The symptom was present temporarily in a large number of his cases during the early part of the first stage, and in fifteen it was persistent, continuing to three days. Of the fifteen thus affected, twelve died, and but three recovered.³ It is sometimes preceded, and may be foreseen by a *guttatim* discharge; but, in many instances, it comes on imperceptibly and without any forewarning. It is sometimes attended with pain about the pubis—a sense of fulness, without any visible enlargement of the bladder, with contraction and contortion of the penis,⁴ and occasionally with an unavailing desire to make water.⁵ But in many, as Savarésy has noticed, the patient experiences no pain at all, and does not appear conscious of the occurrence (p. 282). It has been properly pronounced a perplexing symptom, as nothing has as yet been found to have any effect in stimulating the kidneys to action. The restoration of the secretion must, of course, be hailed as a favourable sign.

The suppression of urine takes place sometimes at an early period, or during the second stage of the disease.⁶ Generally, however, it comes on about the accession of the third. At other times it occurs at a later period.⁷ According to Rochoux, the former is usually observed in the fever of tropical climates, and the latter in the so called yellow fever of temperate regions, and he deduces from the difference, an argument in favour of his opinion respecting the distinct character of the two diseases. But the asser-

¹ Cathrall, on Black Vomit, p. 31, 8th ed.; Rush, iii. 62, 207; Deveze, p. 26; Monges, p. 58; S. Jackson, p. 56; Caldwell, 1805, p. 82; N. O. 1820, p. 10; Ib. 1819, p. 8; Ib. 1839, p. 325; A. Hosack, p. 16; Drysdale, i. 136; Dalmas, p. 9; Girardin, pp. 35, 55; Gros, pp. 10, 11; Cartwright, pp. 11, 13; Lining, ii. 431; Merrill, ii. 222, 3; Ib., ix. 245; Barton, xv. 37, 63; Valentin, p. 174; Townsend, p. 155; Baxter, xxi. 3; Dupuis, xxi. 75; A. Smith, xxv. 502; Thomas, p. 85; Kelly, xv. 378; Harrison, ii. 135; Arejula, p. 160; Palloni, pp. 5, 8; Pym, pp. 223, 4; Fellowes, p. 56; Audouard, p. 61; Proudfoot, xxvii. 250; T. Smith, xxxv. 42; Velasquez, pp. 11, 15; Gillkrest, ii. 271; Pariset, p. 423; Rochoux, p. 510; J. Clark, p. 14; Dariste, p. 135; Vatable, p. 346; Caillet, p. 19; Gilbert, p. 66; Catel, p. 12; Rufz, p. 55; Bally, pp. 244, 283; Maher, pp. 848, 860; Dubreuil, viii. 321, 2, 3; Chisholm, i. 72; Savarésy, pp. 274, 282; Ralph, ii. 67, 74; Moseley, p. 438; Baneroft, p. 14; Pugnet, pp. 336, 360; R. Jackson, Sketch, i. 88, 112, 173; Desportes, i. 199; Barrington, xii. 311; Osgood, p. 13; Evans, p. 257; Dict. des. Sci. Méd., xv. 337; Rochoux, p. 310; Fever on board the Macedonia, p. 29; Moreau de Jonnes, p. 133; Pennell, Med.-Chir. Tr., xxxvi. 251.

² Med. Chir.-Tr., xxxvi. 253.

³ Op. cit. p. 78.

⁴ Chisholm, i. 172, 3.

⁵ Jackson, Sketch, i. 92, 3.

⁶ Chisholm, i. 172; Savarésy, p. 274; R. Jackson, i. 88, 92, 108; Barrington, xii. 311; Rochoux, p. 310.

⁷ Desportes, i. 199.

tion is not corroborated by other testimony. So far from it, we find that in both regions, the symptom varies equally as regards the period of its occurrence, as is acknowledged by M. R. himself, who states that it sometimes occurs in the last stage of prolonged cases of the West Indian yellow fever (p. 311).

The suppression of urine differs in point of frequency in different epidemics. In Rio Janeiro, according to Dr. Pennell, about eighty out of every hundred fatal cases were attended by this symptom.¹ Dr. Fenner thinks that it was much less frequently observed in dangerous cases than formerly, in New Orleans during the epidemic of 1853 (p. 49).

Whether the fatal issue of cases attended with suppression of urine, is always and solely due to, or hastened by, the presence of urea in the blood and serous fluids of the ventricles of the brain—the result of the want of its elimination from cessation of the renal function—has not yet been positively ascertained, few experiments having been made to test the presence of that substance in the parts in question. There is reason to believe, however, that such is the case, judging from the nature of the symptoms observed in instances of the kind, and from the results obtained by Dr. Henderson in the relapsing fever of Scotland,² and by others in typhus fever,³ in both of which great diminution, or even suppression of urinary secretion is apt to occur.

e. RESTLESSNESS AND JACTITATION.—In few forms of disease do we find the patient to exhibit a greater degree of anxiety, restlessness, and jactitation than in the yellow fever. These symptoms may indeed be viewed as characteristic of the disease; for the state of calmness seldom exists, except it be in cases complicated with coma or drowsiness, or in that form of the disease—mostly congestive—marked by general prostration and insensibility. The early stage of fever, during our epidemic of 1793, was characterized by great restlessness, and frequent shifting of posture in search of ease; in the last stage, the restlessness and tossing to and fro became perpetual and distressing.⁴ The same remarks are applicable to our subsequent epidemics,⁵ in regard to one of which (that of 1820) we are told that “it was almost impossible to retain some patients in their beds, from an unconquerable feeling of restlessness with which they were afflicted. No position gave them ease long, and respite from torment was expected from a new attitude or a new resting-place.”⁶ The remarks would apply equally well to the cases observed in 1853 and 1854.

Lining tells us, relative to the fever of 1748 at Charleston, that “restlessness and almost continued jactitation came on the second day.” In the latter stage, the symptom “was surprising; it was frequently scarcely possible to keep the patients in bed, though, at the same time, they did not complain of any anxiety or uneasiness, but, if asked how they did, the reply was ‘very

¹ Med.-Chir. Tr., xxxvi. 252.

² Edinb. Med. and Surg. Journ., lxi. 223, &c.

³ Taylor, Scottish Med. Gaz., i. 289.

⁴ Currie, pp. 21, 24, 25; Cathrall, pp. 24–28; Deveze, p. 28; Barnwell, p. 373.

⁵ Caldwell, p. 83; Firth, p. 26; Pascalis, p. 31.

⁶ Jackson, p. 57.

well'" (ii. 414-420). And a reference to the accounts we have of the subsequent epidemics of that city, as well as of those of other cities of the Union, from Galveston to Boston, will show that the same phenomena have never failed to present themselves.¹

In the fever of Europe, feelings of anxiety and anguish, as also of restlessness and jactitation have not been less conspicuous. Dr. Rochoux, it is true, denies the occurrence, and affirms that patients affected with the disease remain calm and quiet, except in cases attended with delirium, or when, from the onset, the fever is accompanied with acute pain, especially of the gastro-intestinal organs (p. 485). The assertion, however, is disproved by the testimony of writers on even the epidemic of Barcelona, the only one in temperate climates which Dr. Rochoux had occasion to notice; and there is not one on the fever of the rest of Spain, of Gibraltar, and of Leghorn, who has not pointed out these symptoms as deserving of attention.² "Jactitation," says Dr. Gillkrest, "is a remarkable symptom; the patient tosses his head and limbs about incessantly, unable to procure sleep in any position, or relief from the feeling of distress by which he is oppressed."³

Finally, in tropical regions the same phenomena have been noticed in a well-marked and even extreme degree.⁴ They are less prominent, violent, or constant in mild than severe cases—in those who recover, than those who die; though on this subject the rule is not absolute, as some of the most congestive cases are free from them. They attain importance, generally, after the first hours, and are worst between the second and fourth days. Though occurring in cases attended with much acute suffering in the abdominal organs, back, limbs, and head, or with cerebral derangement, and though due in some measure and in some cases to these pathological states, restlessness, anxiety, and jactitation are found to occur even to a great extent in cases

¹ Heustis, p. 112; Brown, p. 12; Addoms, p. 9; A. Smith, *Am. Journ. Med. Sci.*, xxv. 501, 502; Gros, p. 10; Thomas, p. 41; Girardin, p. 55; Barton, p. 10; Ticknor, iii. 227; Baxter, xxi. 37; Randolph, xxiii. 168; Nott, *Charleston Journ.*, iii. 9; Townsend, p. 165; Harrison, ii. 132; Moultrie, p. 6; New Orleans in 1820, p. 9; *Ib.* in 1839, p. 333; Dickson, iii. 254; E. H. Smith, p. 122; Dalmas, p. 10; Valentin, pp. 164-166; Shecut, p. 121; Waring, pp. 46, 47; Bayley, p. 97; Barrington, xii. 312; Kelly, xiv. 379; Drysdale, i. 127.

² Audouard, p. 57; Fellowes, pp. 52, 55; Caisergues, p. 167; Berthe, p. 82; Palloni, p. 5; Proudfoot, xxvii. 231, 251; Pariset, p. 30; Velasquez, p. 12; Gilpin, v. 332; Boyd, p. 299; Pym, pp. 227-233; Arejula, p. 160; Jackson, p. 72; O'Halloran, *Dis. of And.*, pp. 77-123; Copland, iii. 154-158, *Am. ed.*

³ *Cycl.*, ii. 270.

⁴ McArthur, pp. 346, 347; Pugnet, p. 354; Gillespie, p. 39; Moseley, p. 436; Imray, liii. 81; Osgood, p. 10; J. Clark, p. 13; Lempriere, ii. 59-64; Ralph, ii. 70; Savarésy, pp. 270, 280; R. Jackson, *Sketches*, i. 69-92; Rufz, pp. 12, 13; Frost, xiii. 29; Comrie, xiii. 136; Peixotto, i. 413; Maher, pp. 842, 849; Dariste, p. 160; Wilson, p. 8; Dancer, p. 82; Bally, p. 217; Dyott, p. 1003; Caillot, p. 16; Belcher, xxiii. 151; Morgan, *Chapman's Journ.*, iv. 4; Warren, p. 10; Dickinson, pp. 125-128; Rochoux, p. 301; Evans, p. 256; Bruce, p. 278; Hillary, p. 148; Hunter, p. 64; Bone, p. 5; Vincent, p. 23; Jolivet, p. 8; Dupont, p. 19; *Fev. of Cayenne* (1850), p. 175; Catel (1844), p. 225; Copland, iii. 139; Lallemand, *Fev. of Rio* (1850), p. 90.

characterized by different phenomena, and particularly by absence of delirium, and are therefore not necessarily to be referred to the agency of acute pain, or viewed in the light of a manifestation of delirious excitement. In these and other cases they are probably the effect of the feeling of bodily uneasiness resulting from irritation of the ganglionic centres, and are noticed, though generally in a less degree, in all diseases characterized by gastro-intestinal derangement. Though restless and agitated, the patient very usually prefers to lie on his back.¹ He feels easier in that position, suffers less from pain in the hypochondriac region, and breathes with more freedom.

f. WAKEFULNESS.—Similarly conspicuous—and perhaps more so—in the yellow fever of this city, has invariably been the state of wakefulness which, as mentioned in the general description of the disease, is an almost constant attendant on the phenomena last noticed. With the exception, indeed, of cases characterized by coma or stupor, the absence of sleep is an almost invariable symptom of the disease, and proves, as may readily be presumed, a source of the greatest distress to the patient. It is often complete and constant, while at other times it is interrupted by light, short, and imperfect slumbers, which are attended with distressing dreams, and prove more harassing to the patient than even wakefulness itself. In the few cases in which slumbers are more natural and refreshing, the intervals are not marked by a feeling of drowsiness—a circumstance distinguishing the yellow fever from other fevers, the bilious and typhoid, in which drowsiness is very common. It attends often throughout the whole course of the disease; in other instances, during some of its stages. Dr. Rush, in his account of the epidemic of 1793, says: “A coma was observed in some people, or an obstinate wakefulness in every stage of the disease” (iii. 63). Deveze regards the opposite condition as of rare occurrence, and, in speaking of the second stage, remarks: “Sleep, which was interrupted from the commencement, continued in the same state; if, by chance, the patient fell asleep, he was tormented with painful and fatiguing dreams, which rendered his slumber more disagreeable than even the state of wakefulness” (pp. 24, 25). Currie also speaks of the “constant pervigilium” of the first stage, and of the “constant and distressing wakefulness” of the last (pp. 22, 25). It is also noticed pointedly by Cathrall (p. 26), Caldwell (p. 84), and Pascalis (p. 32). Lining long ago stated that “on the first day they generally dozed much, but afterwards were very watchful” (p. 414); and when, in the second stage, “they dozed, their slumbers were but short and unrefreshing (ii. 420). Thomas says that, during the first, second, third, and fourth days, sleep is lost, and speaks of the complete absence of it (*privation absolue de sommeil*) as among the constant and prominent symptoms of the first stage in the yellow fever of New Orleans (p. 83, 1st ed.), where, as well as in other parts of this country—Natchez, Galveston, Key West, New York, &c.—it has been very generally noticed by American writers.²

¹ Rochoux, p. 302; Rouppe, p. 307; Savaresy, p. 274; Bourdon, p. 11.

² Addoms, pp. 9, 10; Barton, pp. 36, 63; Gros, p. 10; A. Smith, Am. Journ. Med. Sci., xxv. 502; Hogg, pp. 412, 416; Ticknor, iii. 225; Townsend, pp. 151–155; New Or-

In Europe, also, as well as in tropical regions, it has scarcely ever failed to attend on this disease, and to prove, as here, a source of much suffering.¹

In some cases it commences from the outset of the attack. More generally, it sets in after the first twelve or twenty-four hours. It is more generally, though not exclusively, an attendant on severe or fatal cases. Pervigilium often gives way, in cases that terminate favourably, to quiet and refreshing sleep; the accession of which, when associated with other favourable symptoms, must always be hailed as a happy omen. In some cases, however, watchfulness has continued obstinate after the total cessation of fever and pain, and even for some days after convalescence has set in.² In other cases, particularly in those which assume a fatal tendency, it glides into stupor or coma, or is replaced by delirium. Though depending, apparently, in some instances, on gastro-enteric inflammation, and at other times on acute inflammatory irritation of the brain and membranes, pervigilium occurs often without being due to such causes—in cases where, the mind being clear and unclouded, we have no reason to suspect the existence of cerebral disease, and where, if gastro-intestinal irritation exist at all, it is not sufficiently severe to occasion such an effect. Besides, the very frequent occurrence of the symptom in question, and the equally frequent absence of cerebral and gastric inflammation, as well as the continuance of the former after the establishment of convalescence, must serve to show that the agency of abdominal or cerebral disease in the production of the phenomenon in question, can only be admitted to be operative in exceptional cases, and not in all, as has been affirmed so far as regards the fever of Europe and this country, by Dr. Rochoux.

This symptom, which differs, in respect to degree, among various individuals during the same season, appears to be more frequent and violent in some than other epidemics. While some writers in this country and the West Indies make no mention of it, or speak of it as of occasional or frequent, but not universal occurrence, and do not represent it as carried to very great or distressing extent, others note it in very different terms. “One of the most constant and distinguishing symptoms of this fever,” says Dr. Blane, “is an obstinate, unremitting, and painful *pervigilium*, which is the more tormenting, as the patient is extremely desirous of sleep. It is seldom that even a *delirium* comes to his relief to make him forget himself for a moment; but

leans in 1820, p. 9; Ib. in 1839, pp. 332, 333; E. H. Smith, p. 122; Stone, p. 553; Moultrie, p. 3; Valentin, pp. 165, 166; Drysdale, i. 127.

¹ Fellowes, p. 202; Audouard, p. 57; Proudfoot, xxvii. 251; Louis, pp. 180, 181; Pariset, pp. 381, 407; Pym, p. 227; Copland, iii. 154–158, Am. ed.; Poissonnière, p. 50; Ralph, ii. 68; Savarésy, pp. 270–280; Frost, xiii. 29; Comrie, xiii. 176; Warren, p. 10; Ruz, 12; Dict. des Sci. Méd., xv. 334; J. Clark, p. 8; Bally, p. 213; Bruce, p. 278; Hillary, p. 148; Osgood, p. 10; Dariste, p. 160; Bancroft, p. 10; Dancer, p. 82; Blane, p. 436; Vatable, p. 345; R. Jackson, i. 68, 105; Rochoux, pp. 304, 305; Vincent, p. 26; Carter, pp. 4, 5; Copland, iii. 139.

² Pariset, p. 381; Rush, iii. 63–207; Stone, vi. 553.

he continues broad awake, night and day, with his reason and senses sound, in a state of the most uneasy agitation.”¹

g. MUSCULAR POWER.—The yellow fever, like other complaints of kindred nature, is often marked by more or less bodily debility, both from the outset and during the progress of the attack. This is more particularly noticed in some modifications of the congestive or adynamic varieties, but is not exclusively limited to these. The patient feels exhausted—every movement is to him a source of labour and difficulty—and as the disease advances, the prostration becomes complete, and the muscular power destroyed to such an extent, indeed, that the limbs appear almost paralyzed. The occurrence of this debility, pushed to a greater or less extent, has been noticed—not in the fever of this country and other parts of temperate climates only, as Dr. Rochoux would wish, for reasons of his own, to have us believe—but in tropical regions also. On this we have the positive testimony of many writers here and elsewhere. Sir Gilbert Blane speaks of the “universal debility” occurring in the first stage of the disease, and of the “extreme muscular debility” of the last” (pp. 405, 415). Dr. J. Hunter tells us that “the voluntary and involuntary motions are equally affected,” and that “the muscular fibre cannot contract with its usual force, and thence a general loss of strength” (p. 144). Pugnet represents the disease as characterized by general debility (p. 353). Dr. Arnold says: “It has been said that the muscular strength is less diminished in this than in other fevers, so that persons afflicted with it have gone about their usual duties till within a short period of death. Of this I do not believe one word; there is no disease wherein the muscular power is so much impaired from the commencement to the termination, as it is in this; particularly if the invasion is brought on by syncope (p. 8). In the first stage of the disease, according to Madrid: “*La fuerza muscular se halla muy debil, y el enfermo estando acostado se cree con fuerza; va a sentarse y prontamente siente que sus musculos no resisten la acci3n en que se les ha puesto, se le vá la cabeza y vuelve á acostarse por necesidad.*” In the second stage: “*Se aumenta la prostracion de fuerzas*” (pp. 6, 24–27). Dr. Ed. Bishop, in a letter to Pym, speaks of the sudden and great prostration of strength in the fever of Guadaloupe in 1795 (p. 118). This asseveration as to the existence of muscular debility in the greater number of cases, and in different degrees from the commencement of the attack, may be confirmed by reference to the writings of Imray (p. 79), Desportes (i. 193–4), Ralph (ii. 66–71), Lempriere (ii. 85), Bancroft (p. 12), Savarésy (pp. 273, 4, 6), Stone (vi. 553), Archer (v. 67), Drysdale (i. 136), Lining (ii. 414, 421), Harrison (ii. 132), Merrill (ix. 245), Dickson (iii. 255), Irvine (pp. 30, 31), C. Drake (xxi. 133), Mabit (p. 11), Leblond (p. 103), Berthe (p. 82), Velasquez (ix. 14), Fellowes (p. 54), Shecut (p. 119), Evans (p. 257), Pariset (pp. 430–1), Wilson (pp. 11, 13), Deveze (p. 25), Levacher (p. 74), Rochoux (pp. 303–4, 503, 541); while others, as Hillary, Fontana, Vatable, Moseley, and Copland, make no mention of the subject; thus leading us to

¹ Diseases of Seamen, p. 436.

infer that a contrary condition of the muscular system of animal life did not fall within the sphere of their observation ; and that the form of disease they saw did not materially differ on the score of muscular strength from other febrile diseases in which the latter is always more or less impaired.

True, however, as it may be that the disease is often characterized by the symptom in question, experience has shown that it as frequently—perhaps more frequently—exhibits throughout its whole course, or during some one of its stages, an opposite condition of muscular strength. There is no disease, indeed, the oriental plague perhaps excepted, in which the latter is more commonly, and to so great an extent preserved. Instances of the kind occurring in the course, or at the close of an attack, are recorded or admitted by many of the very writers who describe the disease as usually attended with prostration. They would be still more frequently recognized, were it not for the fact, that some of the authors cited recognize no difference between the yellow and the common bilious remittent fever, in which muscular prostration is an ordinary symptom, and have, in all probability, attributed to one disease a phenomenon that appertains to another ; while again in some instances the proof of debility is predicated on the occurrence of syncope on assuming an erect or sitting position, which, in truth, was due to some peculiar condition of the cerebral system, and is no way connected with weakness of the muscles. But, be this as it may, the preservation of muscular strength in many cases of yellow fever, is a fact placed beyond the possibility of doubt. It has been found of more or less common occurrence by some, and as very generally present by other writers. In the epidemics of this city, the phenomenon has attracted universal attention. Dr. Rush, after remarking, in his account of the epidemic of 1793, that the disease sometimes came on with syncope and other symptoms, says : “While the muscles and nerves in many cases exhibited so many marks of preternatural weakness, in some they appeared to be affected with preternatural excitement. Hence, patients in the close of the disease often rose from their beds, walked across their rooms, or came down stairs with as much ease as if they had been in perfect health. I lost a patient in whom this state of morbid strength occurred to such a degree, that he stood up before his glass and shaved himself on the day upon which he died.”

Dr. Pascalis, in his account of the epidemic of this city in 1797, says in reference to this matter : “The patient talks much of his expected recovery, and will sometimes even try, with success, to give deceiving proof of it. He wishes to take exercise, and pursue his business. He will get up ; want to dress himself, and will walk very freely. I have seen a few who, on these occasions, were able to sit in company. Every one was astonished, and would remark no other alterations than that of a deep orange colour all over his body, no incoherence in his conversation, and a very confused look. How short, alas ! and deceiving is this last spark of life !” (pp. 36, 37).

There were, in 1797, Dr. Rush states, what Dr. Caldwell happily called walking cases. The patients were flushed or pale ; had a full or tense pulse, but complained of no pain ; had a good appetite, and walked about their

room or houses as if they were but little indisposed, until a day or two, and, in some instances, until a few hours before they died" (iv. 14).

The late Dr. Monges, of this city, who witnessed all our epidemics from 1793 to 1820, inclusive, and saw as much of yellow fever as any physician; and who, withal, was a very acute and profound observer, and distinguished practitioner, regarded the preservation of muscular strength as one of the characteristics of the disease, and as a mark of distinction between the latter and the bilious remittent (p. 57); and most of our writers have dwelt to a greater or less extent on this phenomenon;¹ examples of which were offered during the epidemic of 1853, as well as among the sporadic cases of the year following.

Nor has it been less noticed in other cities of the Union and elsewhere—in New York,² Baltimore,³ Norfolk,⁴ Middletown,⁵ Savannah,⁶ Pensacola,⁷ Key West,⁸ New Orleans,⁹ Galveston,¹⁰ Natchez,¹¹ Mobile.¹² In Spain¹³ and the West Indies,¹⁴ the phenomenon has been frequently observed. In many cases, the muscular energy discloses itself by the freedom of motion enjoyed by the patient while in a recumbent position, by the facility with which he changes his position—a facility which the feeling of restlessness he experiences leads him to exercise almost incessantly. It is still more evidently shown by the force which is oftentimes required to control the patient and keep him in bed. Often he sits up in bed, or on a chair; gets up without assistance to use his chamber or for other purposes; walks about his room; dresses, shaves, goes down stairs, or even walks into the streets, as if in a state of convalescence; and continues so to do until a few moments before expiring.

Already, in the chapter on Black Vomit (p. 284), I have related several cases of the kind.

Dr. Jourdain, speaking of the symptoms observed at *Port du Passage*

¹ Currie, pp. 26, 28; Pascalis, pp. 36, 7; Caldwell (1826), p. 159; Ib. (1805), p. 84; Klapp, iv. 85; Chapman, ix. 130; Ffirth, p. 26; Barnwell, p. 232.

² Smith (E. H.), p. 123; Townsend, p. 166.

³ Drysdale, i. 130; Jameson, vi. 448.

⁴ Archer, v. 67.

⁵ Tully, p. 299.

⁶ Waring, pp. 46, 55.

⁷ Barrington, xii. 312.

⁸ Ticknor, iii. 225.

⁹ Gros, pp. 9, 11, 12; Baxter, xxi. 3; Duprey de Chambery, xxi. 17; Thomas, pp. 85, 6; N. O. 1819, p. 9; Ib. 1839, p. 334; Girardin, pp. 25, 6; Dowler, Fev. of 1853, p. 63.

¹⁰ A. Smith, p. 501.

¹¹ Cartwright, ix. 10, 12; Merrill, Memphis Recorder, iii. 166.

¹² Kelly, xiv. 379, 380; Lewis, N. O. J., i. 416.

¹³ Dict. des Sci. Méd., xv. 335; Jourdain, Ann. de la Méd. Phys., iv. 535, 541, 543; Jackson (R.), Spain, pp. 62, 79, 83, 87, 96; Lining, ii. 198, 200; Gillkrest, ii. 272; Audouard, pp. 57, 61, 63; Pariset, pp. 379, 430, 525; Fellowes, p. 54; De Maria, p. 70.

¹⁴ Dict. des Sci. Méd., p. 335; Jackson, Tr., pp. 258, 9; Ib., Sketch, i. 69; Pinckard, ii. 82, 3; Gillespie, p. 41; Bally, pp. 222, 223, 224; Chisholm, i. 154; Dariste, pp. 136, 152; Frost, xiii. 32; Comrie, xiii. 176; Steward, Med. Reg., iii. 187; Lempriere, ii. 64; Blair, p. 65; Rochoux, pp. 304, 548; Anderson, p. 10.

(Spain), in 1823, remarks that the extreme and rapid prostration of the vascular system did not harmonize with that of the muscular system. "Indeed, the latter, in a great number of cases, remained perfectly unimpaired to the last moments of life. Thus, individuals were seen who, though cold as ice, without pulse, and exhibiting all the symptoms indicative of the approach of death, rose without assistance from their beds and walked with a firm step to the water closet."¹

Dr. Cartwright relates, that he knew some patients in the Natchez Hospital at the close of the disease, "suddenly acquire a great accession of strength, arise from their beds, get hold of brooms, or some other such things, and parade the rooms, beating and blustering about to the great terror of the whole house until their death, which took place suddenly."² In 1819, at New Orleans, instances are reported of persons labouring under the worst symptoms of the disease, calling on their physicians for advice (*Baxter*). It is not uncommon to find individuals complaining of slight or no indisposition, walking about and attending to their usual avocation, and exhibiting to all, perhaps, but a practised eye, no indications of danger, and yet soon sinking with black vomit, coma, convulsions, &c. Instances of the kind have been recorded by almost every succeeding writer from the days of Labat to our own, and from the entire preservation of the muscular power, have received the name of walking cases.³ Dr. Kelly relates several cases of the kind. "A Frenchman complained of being unwell. He consulted a medical friend, one of his own countrymen, to whose residence, about a mile distant from his own, he walked every day. On the fourth day of his attack I was called to him, and the first thing that met my eye was the black vomit. Coma and death soon followed."

Dr. Merrill, formerly of Natchez, now of Memphis, Tennessee, relates the following interesting case: "In 1823, I was summoned to the wife of Mr. Jonathan Thompson, who had just been moved some three miles from the city (Natchez), upon the first alarm of the epidemic. Mr. T., whom I had never before seen, received me upon a gallery, and while awaiting for a few moments for the patient to invite me in, I observed, to my great surprise,

¹ Ann. de la Méd. Phys., v. 260.

² It may not be improper to remark, in corroboration of these statements, that in the writings of some of those who contend for the constant loss of muscular strength, facts may often be gleaned of a kind calculated to establish a contrary view. It has been seen that Dr. Arnold does not believe one word of what has been said about the power of the muscles being less diminished in the yellow than in other fevers. He expresses this at page 8. At page 10, he says: "It is not uncommon to see a patient sit up (in the last stage), ask for food, attempt to shave himself, and wish to quit the room; a marked peculiarity of the disease, showing, as it were, a return of the mental faculties previous to death." Others would presume that it showed likewise a return of the muscular powers. How often have such things been seen in other fevers?

³ Labat, ii. 75, 6; Desportes, i. 41; Pinckard, ii. 82, 3; Dict. des. Sci. Méd., xv. 335; Caldwell, p. 84; Kelly, xiv. 378, 9; Rush, iv. 14; Deveze, p. 28; Jackson, i. 358; Townsend, p. 151; Monges, p. 62; Bally, p. 223; Harrison, ii. 133; Merrill, Memphis Med. Rec., iii. 166.

that in the intervals of conversation, he gulped up whole mouthfuls of black vomit. I intimated to him, that he himself appeared to need assistance; but he repelled me in a decided manner, and said: 'I wish you only to prescribe for my wife.' I sought out his nephew, who was an inmate of the family, and told him his uncle had but a few hours to live, and if he wished to make his will he had no time to lose, but in two or three hours time he would be incapable of doing it. A friend was sent for from the city; but it was too late. He was demented on his arrival, and remained so until he died, discharging all the while immense quantities of black vomit."

Labat tells us of a missionary who complained only of a slight headache; but had a good appetite, and was about eating his supper when the physician came in. The latter, having examined and felt his pulse, sent him to bed, informing the superiors that the disease would end fatally before noon of the next day. He died at 9 A. M.¹

There was great variety in muscular power in different persons, says Dr. E. H. Smith, of New York, speaking of the fever which prevailed in that city in 1795: "A man, who died with the very worst symptoms of the fever, the evening before his death, rose from his bed, ran down two flights of stairs, returned, and was only prevented from going down a second time by his nurse having locked him in his chamber."²

Dr. Townsend refers to a case "where the patient continued to walk about for several days as though he was in perfect health; the stomach and other functions all regularly performing their duty until black vomiting suddenly came on, and in a few hours carried him out of the world" (p. 151). The same physician relates the case of an individual who dressed himself, put on his overcoat and hat, and though his laborious inspirations, livid, bloated face, and cold extremities showed that he was actually dying, continued to grope about the room, and was even, to the last hours of his life, with difficulty restrained from going into the street. He repeatedly went to the door, to the astonishment of all the neighbours, saying that he wished to breathe the fresh air and to go home. Another patient, who had black vomit to a profuse degree, got up an hour before his death, said nothing was the matter with him, and dragged his bedstead across the room to the window.³

It is not necessary to inquire here into the nature of these cases. They are referred to as illustrating the fact of a complete preservation of the muscular strength amid circumstances indicating an advanced degree of disorganization of important organs. In such cases, the disease has evidently proceeded through its several stages, though in an insidious manner, and while the poison has overwhelmed the powers of life in many particulars, and

¹ I had barely finished writing the above paragraph before I was requested to see a case of yellow fever—the first reported this season (1854). The symptoms were well-marked, and the patient died on the night of the fourth day with black vomit, jaundice, &c. She retained her muscular strength in a remarkable degree from beginning to end. A few hours before death, she walked with little assistance to the privy, and, five minutes before expiring, rose by herself, sat on the close stool, and walked back to bed.

² Fever of New York in 1795, Webster's Collection, p. 123.

³ Ibid., pp. 166, 7.

thereby laid the foundation of inevitable death, it has scarcely touched some of the organs of animal life, and among these the muscular system.

The phenomenon is more generally noticed in some forms of the disease than in others. It is rarely, if ever, present in the congestive or adynamic form, and is generally more marked in degree in cases of a different kind, in which the vascular reaction is feeble. Lempriere, for example, found his hybrid form of the disease, which corresponds to the adynamic or congestive form of other writers, to be characterized by considerable debility, while in the ordinary form of the fever, the muscular strength seemed unimpaired (ii. 64, 85). In some instances, however, patients affected with malignant yellow fever exhibit, from beginning to end, much greater muscular strength than is found in other fevers characterized by a kindred condition of the vital powers. In many instances, it is exhibited in all the stages of the disease; in other cases, the patient, after possessing considerable strength in the early period, loses it as the disease advances; while, in another set of cases, even of the malignant form, the debility attending the first and second stages gives way in the last to an opposite condition of the muscles, and the patient, who, a few days or hours before, lay prostrate in his bed, is now enabled to rise and to walk about as if in a state of convalescence or health. Finally, the preservation of muscular strength appears to be more frequently observed, and to exist in a more marked degree, in some epidemics than in others. Arejula remarks that, during the fever of Malaga in 1803, patients suffered more from the prostration of the animal powers than had been the case at Cadiz three years before.¹ Bally, who saw the disease in St. Domingo in 1802 and 1803, and in Barcelona in 1821, remarks that on the latter occasion the muscular energy, though evident, was not as well marked as it had been on the former.² It is, probably, from his having observed a similar difference (which, true to his usual mode of proceeding, he exaggerated) between the fever of Barcelona and that which he had noticed in Guadaloupe some years before, as well as from his ignorance of the statements of other writers, that Dr. Rochoux was led to found on the condition of the muscular system an argument in favour of the dissimilar nature of the disease as it presents itself in tropical and temperate regions (pp. 547, 548).

CHAPTER XVI.

NERVOUS SYSTEM.

DELIRIUM.—The condition of the intellectual faculties in the yellow fever is a subject fully deserving our consideration. In the following inquiry, I shall note—1st, the frequency of the derangement of those faculties; 2d, the

¹ Edinb. Journ., i. 449.

² Pariset, p. 525.

period of the disease at which it occurs, and the course it pursues; and 3d, the particular forms it assumes.

1. *Frequency*.—From the foregoing description of the disease, the reader will perceive that delirium, so far from being an habitual and constant symptom, and constituting anything like an inherent or necessary part of the disease, fails, in a large number of cases, to manifest itself, not only during some part, but during the entire course of the attack. That facts and statements tending to invalidate this statement are on record—that delirium often appears early, and continues throughout—and that still more frequently it shows itself, under various circumstances, at a late period of the attack, no one acquainted with the disease can deny. Dr. Rush, in his description of the fever of 1793, says: “A delirium was a common symptom” (iii. 64). In speaking of the yellow fever of temperate regions—or, rather, of that of Barcelona, the only one of which he had any personal knowledge—Rochoux remarks that delirium is a truly habitual symptom, and is present in almost every case—none of those who die, and few of those who recover, being free from it (p. 499, 570, 571). By a reference to other writers on the yellow fever of various countries, it will be found that the symptom, under similar circumstances, is of not unfrequent occurrence.¹

But, however this may be—whatever may have been the habitual occurrence of delirium at some epidemic seasons and in some particular localities, and admitting that everywhere and under all circumstances it often manifests itself at some period or another of the disease, a reference to the best authorities, and to observations made here, will bear me out in the opinion expressed, and will even go to show that the integrity of the mental faculties may not unreasonably be regarded as a peculiar characteristic of the disease; that, taking a wide survey of the subject, instances in which delirium appears early and continues throughout must be viewed as exceptions to a general rule; that it forms no part of the pathognomonic phenomena of the disease; that it is, in fact, nothing more than a complication, which may or may not exist without affecting the character and nosological position of the cases observed; and that, in the majority of cases in which it appears, it does so—agreeable, indeed, to the statements of even many of the afore-mentioned writers—in some forms only of the disease, and in these only at a period of the attack corresponding to that in which, in the most dissimilar complaints, the brain usually becomes implicated. Dr. Rush, after having stated, as already seen, that in 1793 delirium was a common symptom, adds: “Many, however, passed through the whole course of the disease without the least derangement of their ideas, even when there were evident signs of a morbid congestion in the brain”

¹ Villasca, quoted by Rochoux, p. 499; Addoms, p. 9; Bancroft, p. 10; Hillary, p. 149; Chisholm, i. 152; Arnold, p. 8; Lining, ii. 415; Lallemand, pp. 91, 92; R. Williams, p. 494; Dickinson, p. 132; New Orleans in 1839, p. 325; Fenner (1853), p. 49; Caisergues, pp. 121, 122; Morgan, Med. and Phys. Journ., iv. 4; J. Clark, p. 8; Ffirth, p. 26; Warren, pp. 9, 15; Pascalis (1797), p. 36; Comrie, xiii. 175; Hunter, p. 70; Frost, xiii. 33; Belcher, xxiii. 250; Hogg, i. 413; Finlay, p. 16; De Maria, p. 70; and other writers in this country and elsewhere.

(iii. 64). And this will be found to have occurred even in cases that terminated in death. In his account of the epidemic of 1794, the same author says: "Delirium was less common than last year" (iii. 208). In 1797, "the mind was seldom affected by delirium after the loss of blood" (iv. 11). In 1799, "delirium was less common in adults than in former years" (iv. 57). While on the subject of the symptoms of the first stage of the fever of 1793, Dr. Cathrall says: "The intellectual faculties at times appeared confused, but seldom a complete delirium. If delirium appeared, it was in the last stage" (pp. 25-28). Currie, too, remarks: "Some confusion of the intellect, attended with constant pervigilium; but seldom so much derangement of the reasoning faculties as to amount to violent delirium." In the last stage, some of his patients had a cheerful delirium, but in many the mind was not apparently affected (pp. 22, 28). Other writers, who have described these epidemics—Deveze (p. 29), Barnwell (p. 372), Nassy (p. 20)—have left on record ample proofs of the fact that, though some patients became more or less delirious at some stage of the disease, a large number, if not a majority, went through without a notable derangement of the mental faculties. Even Dr. Pascalis, who figures above among those who regard delirium as a common symptom, admits that in 1797 it frequently occurred that patients retained their reason till the last moment (p. 36). In 1805, delirium, though an occasional, was by no means a common symptom in the early stages.¹ In 1820, many who laboured under the worst form retained their senses to the last; in others, there was a state of reverie, or an appearance of great distress.² In the mildest form, there was only a confusion of mind. And in 1853, delirium, during the early stage particularly, was not common. Taking all the cases conjointly, we may say that as great a number, in all our epidemics, went through the disease, whether to recovery or death, without delirium as with it. So common, indeed, is the integrity of the mental functions in our yellow fever, that the late Dr. Monges, of this city, than whom few individuals saw more of the disease, considers the absence of delirium as one of the distinguishing marks between the latter and our common bilious remittent fevers, in which the mind usually becomes involved in the derangement of the system.³

Nor is this frequent absence of delirium peculiar to the yellow fever of Philadelphia. In other parts of the United States, a similar observation has been made, every writer speaking of it as a usual or frequent occurrence; admitting positively, or allowing us, by their silence relative to that symptom, to infer that delirium, properly speaking, occurs only occasionally in the early stages, or representing it as doing so only at the close of the disease.⁴

¹ Caldwell, p. 84.

² Jackson, p. 57.

³ N. Am. Med. and Surg. Journ., ii. 57.

⁴ Dalmas, pp. 10, 11; Townsend, p. 146, 155, 158, 168; Thomas, p. 83; Valentin, p. 169; Girardin, pp. 34-56; Merrill, ix. 244; Cartwright, ix. 12; Dickson, iii. 256; N. O. 1839, pp. 332, 334; Harrison, ii. 133; Bayley (1795), p. 93; Moultrie, pp. 3, 5; Shecut, p. 121; Davidge, p. 104; Stone, vi. 555; Hill, v. 90; Archer, v. 67, 68; Barton, p. 10; Waring, p. 46; Warren (in Tytler), p. 502; E. H. Smith, p. 118; A. Hosack,

In the fever of Europe, whether at Cadiz, Malaga, Gibraltar, Leghorn, Minorca, or Marscilles, the same frequent, if not usual integrity of the mental faculties—the same limitation of delirium, when it does occur, to the after periods of the disease—has been noticed.

Contenting myself with referring to the writings of Arejula (p. 160), *Ib.*, *Ed. J.* (i. 448), Fellowes (li. 6), Caisergues (p. 170), Berthe (p. 87), Boyd (pp. 299–301), Copland (iii. 141), Jourdain (v. 259), T. Smith (xxxv. 42), Barral (p. 19), Dufour (iv. 51, 52), and O'Halloran (p. 76), I shall dwell on a few statements which establish the fact beyond the possibility of doubt. Palloni (p. 5) says: "In mezzo di sì gran tumulto le faculte mentale si conservano libera ed entiere." Pym makes no mention of delirium in the description of his first, second, and fourth varieties, and we may presume that it did not exist (pp. 225–231, 233, 234). Louis does not appear to have noticed much delirium at Gibraltar in 1828. Of fifteen fatal cases, seven had none at all. The others were only slightly delirious, and in most instances only during the last twenty-four hours. Even under such circumstances, the derangement was slight, consisting in an incoherence and a confusion of ideas (p. 182). The same result obtained among the cases communicated to the commissioners. In those who recovered, Louis found *no* delirium (p. 193). He, therefore, classes that symptom among the adventitious ones (p. 189). Gillkrest speaks of the uniform integrity of the cerebral functions in the first stage, and the extremely frequent integrity of these to almost the last moments of existence in the worst and most fatal forms of the disease (p. 278); and seems to admit the existence of delirium only in some cases of a protracted form (p. 273, note). Even as regards the fever of Barcelona, where, as we have seen, Rochoux found delirium of usual occurrence, other observers have arrived at very different results. Pariset calls attention to the preservation of the intellectual faculties in the first and second stages, and sometimes throughout; while of the delirium of the third stage he remarks, that it was simply an occasional aberration of ideas (pp. 381, 403). And Audouard is still more explicit on the subject, admitting, as he appears to do, the supervention of delirium only when the patient is almost in *articulo mortis*, and even then not always, for he remarks that it is not rare to find patients retaining full command of their intellectual faculties up to the very last moments of life (p. 63).

In hot climates, too, the very common absence of delirium in the early stages—nay, even the frequent perfect integrity of the cerebral functions throughout the whole course of the disease—and the limitation of the symptom, when it does occur, to the closing stage, may be mentioned on the authority of numerous writers, who either make the statement in positive terms, or leave us to infer the fact in question from their silence relative to the condition of the intellectual functions.¹

p. 15; Drysdale, i. 128; C. Drake, xxi. 124; Tully, p. 299; Willey, Repos., vi. 125; Kelly, xiv. 377–8; Gros, pp. 10–13; A. Smith, xxv. 502; Randolph, Med. Repos., xxiii. 169; Cooke, N. O. J., x. 645; Wragg, x. 81.

¹ Evans, p. 241; Bruce, p. 278; Rufz, p. 13; Dickinson, pp. 126, 132; Fontana, p. 73; Pugnet, pp. 353–7; Caillot, pp. 19, 20; Th. Clark, p. 8; Barrington, xii. 311;

Rochoux, who thinks that delirium is a usual attendant on the yellow fever of temperate climates, regarded it as never present in simple cases of the fever of tropical regions, and, when occurring, as being always the result of a complication (pp. 312, 570). "In no acute disease," says Bally (p. 211), "do the intellectual faculties preserve so well their integrity as in the present, and especially the one of which I am giving the description (the epidemic of St. Domingo in 1802 and 1803), and in which I have seldom observed delirium." According to Dariste, the "integrity of the intellectual faculties constitutes an essential characteristic of the disease, when the latter is not complicated with inflammation of the brain" (p. 136).

Delirium appears more frequently in some seasons than in others. In New Orleans, during the epidemic of 1819, it showed itself in a greater number of cases than it had done two years before.¹ In 1853, too, the head symptoms were more common and dangerous than in former epidemics.² The same diversity has been noticed in the West Indies;³ and we have seen, on the authority of Dr. Rush, that such was the case also in this city.⁴ This greater tendency to delirium during some epidemical seasons, prepares us to expect a corresponding difference, in point of frequency, in the various forms of the disease; which difference in frequency of those forms is, as is well known, under the influence of particular seasons. It has been found to prevail more readily in that form described under the name of ataxic (*Harrison*, p. 134; *Pym*, p. 233; *Madrid*, p. 30), or in the inflammatory (*Wilson*, p. 8; *S. Jackson*, p. 57); while in the congestive or malignant it is generally wanting (*Archer*, p. 68; *Gillkrest*, p. 278; *S. Jackson*, p. 57); and in the mild form it seldom occurs (*T. Smith*, xxxv. 42; *Louis*, p. 193; *S. Jackson*, p. 59). Nor does delirium fail to present various modifications in the several grades or forms of the disease. In mild attacks, it amounts generally to little more than a confusion of mind, or stupor and slight wandering, or a low muttering.⁵ In the inflammatory, as well as in some varieties of the malignant, it is maniacal.

In not a few instances, the delirium, after having prevailed during a greater or shorter period, subsides at the last moments of life; and the patient, who, some moments before, was perfectly out of his mind, dies in full possession of his faculties, or loses them only a few moments before death.⁶

2. After what has been said above, it will not be necessary to dwell long on the subject of the period of the disease at which the cerebral functions become disturbed. In some instances, delirium occurs early and during the stage of reaction, and subsides during the remission, not to reappear, except

Hillary, p. 149; Imray, liii. 81; Peixotto, i. 413; Moseley, pp. 436-8; Bancroft, p. 12; Jackson, Sketch, i. 66, 73, 91; McArthur, p. 346-8; Savarésy, pp. 275-6, 8, 283; Ralph, ii. 67-72; Jolivet, pp. 9, 11; Anderson, p. 5; Bourdon, p. 11; Holliday, p. 9; Leprieux, ii. 60-2; Dict. des Sci. Méd., xv. 339, 340; Maher, p. 844; Guyon, p. 85 (note); Gilbert, pp. 48, 66; Blane, p. 409; Gillespie, p. 41; Vatable, p. 346; Madrid, p. 30; Dariste, p. 136; Makittrick, p. 4; Ferguson, Recol., p. 146; Joubert, pp. 966-8.

¹ See Rep. to Med. Soc. (1819), p. 36.

² Fenner, p. 49.

³ Dict. des Sci. Méd., xv. 334-340.

⁴ Works, iii. 208, iv. 11, 57.

⁵ Frost, xiii. 33; Ralph, ii. 72; S. Jackson, p. 61.

⁶ Hillary, pp. 149-152; Arnold, p. 9; Evans, p. 242; Ffirth, pp. 27, 28.

occasionally at the approach of death;¹ or it continues throughout. In most cases, however, it is only an attendant on the last stage of the disease, or appears at the closing scene. In a few cases, it persists for a greater or less length of time after the recovery of the patient, or shows itself at the opening of the convalescent state.²

3. The delirium which occurs in the yellow fever assumes various forms, according to the idiosyncrasy of the patient, the period at which it shows itself, the peculiar form assumed by the disease, and other modifying influences, which it would take too long to enumerate.

A. "The delirium attending the malignant pestilential fever," says Dr. Chisholm, "is of a peculiar cast. During it, the countenance, the eyes, and the actions of the patient resemble very much those of a person inebriated." "It is almost always mild, and never furious" (i. 152). The same remark was made at Grenada, on the same occasion, by Dr. Steward.³ It has been noticed also, in this country, by Drs. Merrill (ix. 244), Cartwright (ix. 12), Hogg (p. 414), and Townsend (p. 156). Dr. Merrill remarks that, in some forms of the disease (the inflammatory), there were cases that commenced with great exhilaration of spirits, constant talking, and singing. In this city, Dr. Currie (p. 22), and in the West Indies, Dr. R. Jackson (p. 47) and Rochoux (p. 315) noticed the form of delirium described by Chisholm. It has also occurred in our later epidemics. Dr. Townsend has called attention to the circumstance that, in some cases, the patient appears to be under the dominion of a delusive train of ideas, and a fatal confidence that he has recovered from his illness, and is about to resume on the morrow his wonted occupations. "In others, the hallucination changes to a particular species of derangement, and the patient gets up out of bed, dresses himself, and says he wishes to go home. He does not seem to recognize his nearest relations, and loses also the consciousness of his own personal identity, thinking that some other individual is talked of when his own name is mentioned, and joining in the conversation as though he were a third person."⁴

B. In other instances, the delirium assumes the form of melancholy. The patient is struck with the idea of his approaching end, making it the constant theme of his discourse.⁵

C. In a different set, the patient appears as in a state of hebetude, of apathy, or automatic languor, accompanied with somnolency and incoherency of ideas; or he is taciturn, and, though comprehending what is said, refuses to answer.⁶

D. It is sometimes characterized by a state of reverie, and, when spoken to, the patient starts with surprise, and answers in a hurried manner.⁷

E. In many cases, the delirium of the yellow fever assumes a character of

¹ Dickson, p. 256; S. Jackson, p. 59; J. Clark, p. 8; Thomas, p. 83; Lining, ii. 418; Audouard, p. 56; Copland, iii. 141; Wragg, x. 81.

² Rush, iii. 65; Rochoux, p. 501; Pariset, p. 438; Laso, &c., quoted by Rochoux.

³ Med. Repos., iii. 187.

⁴ Works, i. 156.

⁵ Rochoux, pp. 314, 315; J. Clark, p. 8.

⁶ Rochoux, pp. 314, 499; Boyd, p. 300; Waring, p. 46.

⁷ S. Jackson, p. 57; Rouppe, p. 306; Rochoux, p. 313; Townsend, p. 156.

a peculiar kind. Unlike what takes place in remittent fevers, in which there is a rapid and unconnected succession of ideas over which the patient can exert no control, there are, in the disease under consideration, some fixed ideas deeply impressed on the mind, and which the patient has the power of retaining before him, or which, apart from consciousness or the will, remain fixed in the mind. Dr. Imray, who has called attention to this fact, and whose words I have copied, says that, reasoning from these impressions, the patient comes to correct conclusions. The subjects upon which he dwells are usually his ordinary occupations and pleasures. "He imagines, for example, that some of his relatives at a distance are ill, and that he must visit them without delay. He issues all the necessary orders preparatory to a journey, and, if permitted, will leave his bed and proceed to dress."¹

F. In some cases it is of the low, muttering kind.²

G. In another class of patients, there is only a torpidity, confusion, or loss of memory. This principally occurs in mild cases, or in the early stage of the disease.³

H. In a different set, again, the patient becomes violent, ungovernable, and, as it were, maniacal, accompanied at times with an absence of fever. Such was the degree of this mania, in one man mentioned by Dr. Rush, "that he stripped off his shirt, left his bed, and ran through the streets, with no other covering than a napkin on his head, at eight o'clock at night, to the great terror of all who met him" (iii. 64, 65). Dr. Rush observes that the symptoms of mania occurred most frequently towards the close of the disease (*ib.*). "In the cephalic cases," says Dr. Wragg, "the delirium, frenzy, and mental alienation continue and increase till the strait-jacket alone can restrain the violence of the maniac, or till the oppressed brain can no longer supply the nervous prolongations with the required amount of irritability."⁴

I. In some, there may be a simple aberration of intellect or perturbation of ideas.⁵

K. In many cases—sometimes early, more generally in the latter stages—the mind falls into a state of stupor.⁶

¹ Edinb. Journ., lxiv. 336.

² Kelly, xiv. 378; Ralph, ii. 72; Arnold, p. 8; Evans, pp. 241–256; Currie, in Carey, p. 13, 4th ed.; Townsend, p. 155; Bancroft, p. 31.

³ S. Jackson, p. 61; Currie, p. 22; Louis, p. 182; Moseley, pp. 436–438; Rufz, p. 13; Kelly, xiv. 377; Willey, vi. 125; Waring, p. 46; Cathrall, pp. 25–29; Randolph, Med. Repos., xxiii. 169.

⁴ S. Jackson, p. 57; Waring, p. 46; C. Drake, xxi. 124; Wragg, x. 81; Fellowes, p. 51; Arejula, i. 448, 449; R. Jackson, Sk., pp. 50, 57, 58; *Ib.*, Fever of Spain, pp. 73, 84, 156; De Maria, p. 70; Finlay, p. 16; Dict. des Sci. Méd., xv. 339; O'Halloran, p. 85; Ralph, ii. 72; Arnold, p. 8; Madrid, p. 30; Comrie, xiii. 175; J. Clark, p. 8; Kelly, xiv. 378; Wilson, p. 9; Rochoux, pp. 313, 500; Bancroft, p. 12; Hunter, pp. 65–67; Moseley, p. 433; Savarésy, p. 276; Poissonnière, p. 50; Bally, 233.

⁵ Pariset, p. 403; Holliday, p. 9; Boyd, p. 300; Currie, Fever of 1797, p. 219; Rochoux, p. 499; Louis, p. 182.

⁶ C. Drake, xxi. 124; Kelly, xiv. 378; S. Jackson, p. 59; Arejula, i. 448; Evans, pp. 241, 256; Rochoux, pp. 313, 419; Audouard, p. 56; Stone, vi. 555; Barral, p. 19; Boyd, p. 300.

L. In as many more—at times early in the disease, more frequently at an advanced period—confirmed coma sets in, and, in fatal cases, follows the patient to the close of life—following on delirium, or alternating with it.¹

M. In very many instances, the patient, though seemingly in perfect possession of his reasoning powers, and noticing accurately, and conversing sensibly with his friends and attendants about everything surrounding him, to which his attention is called, yet exhibits a peculiar state of the intellectual faculties, which may, without impropriety, be classed under the head of delirium. He will not, and cannot be made to believe that he is dangerously ill. However well acquainted he may have been when in health, with the usual course of the disease, and whatever may be the fatal tendency of the symptoms under which he labours, he persists in the assertion that he is getting well, and even when on the brink of the grave, maintains with confidence that he will soon be out and about again. Dr. Imray cites in illustration the case of an officer who, when the black vomit was pouring from his mouth, said: "It was nothing but a little biliousness." Not many hours after, he expired.² Speaking of the closing moments of life, Dr. Blair says: "The pulse loses strength, till at last it ceases to be felt at the wrist, and the patient dies with intelligence unclouded and his muscular strength but little impaired, telling you he is getting quite well, or as a poor dying sailor expressed himself, 'illigant, this mornin'' (pp. 65, 6).

This peculiar distortion of judgment has been noticed here in 1853, as well as in our former epidemics (*Jackson*, p. 58). It is mentioned by various writers.³

N. Frequently, in the early part of the attack the faculties are in a state of high excitement without decided aberration. The patient, exceedingly watchful, listens with great anxiety to what is said around him, to ascertain opinions as to his condition; questions others on the subject. When danger exists, though reluctant, at first, to believe that he has the disease, as soon as he feels convinced of it, he gives way to the greatest despondency and alarm.

This state of the intellectual faculties, even before actual delirium occurs, frequently gives way, as the disease advances, to one of utter indifference, or to a confident hope of recovery, even when the symptoms are mortal. The same feeling of apathy and insensibility to danger, or to any external impression, sometimes presents itself in the most malignant varieties from

¹ Bruce, p. 278; Kelly, xiv. 378; Barrington, xii. 311; Dufour, iv. 51, 52; Moultrie, p. 5; Chisholm, p. 154; Moseley, p. 438; Fontana, p. 73; Addoms, pp. 10, 11; Lining, ii. 425; Ralph, p. 72; Rufz, p. 13; Frost, xiii. 29; Caisergues, pp. 121, 122, 170; Currie, Letter to Dr. Center (in Carey); *Ib.*, Bil. Fev., p. 219; Savarésy, p. 273, 277; A. Hosack, p. 15; Townsend, pp. 146, 155; Bancroft, p. 31; Hunter, p. 70; Valentin, p. 170; E. H. Smith, p. 117; Tully, p. 299; Caillot, p. 20; Dyott, p. 1005; Wilson, p. 11; Monson, p. 180; Berthe, pp. 87, 88; Smith, xxxv. 42; Rufz, pp. 13-15; Dickinson, p. 132; Wragg, x. 81; Archer, v. 67; Harrison, ii. 134; Boyd, p. 301; Palloni, p. 5; Finlay, p. 16; Anderson, p. 6; Jourdain, v. 259.

² *Edinb. Journ.*, lxiv. 336, 7.

³ Fever on board *Macedonia*, p. 29.

the commencement. This was observed by Imray, in Dominica, during the epidemic of 1838 (liii. 84).

Convulsions and Spasms.—Delirium is not the only phenomenon connected with the nervous centres which presents itself in the yellow fever. Patients, in some cases, become affected with convulsions, either general or partial. Dr. Rush, in the epidemic of 1793, noticed “convulsions in every part of the body” (iii. 63; iii. 71); or in all the limbs; or, again, in only a part of them (iii. 64). “In some, a violent cramp, both in the arms and legs, attended the first attack of the fever. I met with one case in which there was a difficulty of swallowing, from a spasmodic affection of the throat, such as occurs in the lockjaw” (iii. 64). Convulsions were not uncommon in 1797 (iv. 12), and in our other epidemics. Elsewhere they have been noticed in a variety of cases, both in the form of general convulsions,¹ or spasms or cramps of particular muscles, limbs, face, jaw, lips, tongue, eyes, &c.²

Convulsions of the general kind, in the very large majority of cases, occur in the latter stage, and even during the closing moments of life. In some instances, however, they appear early, and from the commencement of the attack.³

Though occurring generally in cases attended with derangement of the intellectual functions, instances are sometimes encountered, in which they are unconnected with such a derangement, and recur at shorter or longer intervals.⁴

Occurring only in a limited number of cases—the large majority going through the attack to recovery or death without experiencing them—convulsions cannot be viewed as forming a necessary part or characteristic symptom of the fever, and should probably be held in the light of a complication depending on some particular and accidental morbid condition of the cerebral centres—or on some peculiar idiosyncrasy of the patient, on his period of life, his habits, &c. (*Rochoux*, p. 556). They are more common and dangerous in some epidemics than in others (*Fenner*, p. 49).

¹ Addoms, p. 11; Drysdale, i. 30; Archer, v. 68; Tully, p. 299; Wragg, x. 81; Valentin, p. 169; Girardin, p. 56; Harrison, ii. 134; Moultrie, p. 7; Lining, ii. 425; Stone, vi. 555; Kelly, xiv. 378; Audouard, p. 64; Fellowes, p. 55; Proudfoot, xxvii. 250; Palloni, p. 6; Caisergues, p. 179; Th. Smith, xxxv. 42; Velasquez, p. 12; Pariset, Obs., p. 30; Boyd, p. 301; Jackson (Spain), 73, 84, 5; Jourdain, v. 259; Lempriere, ii. 66, 80; Hunter, p. 69; Hillary, p. 150; Savarésy, p. 277; Chisholm, i. 150, 1, 5; R. Jackson, 59, 75, 154; Ralph, ii. 72; Frost, xiii. 32. Comrie, xiii. 177; Peixotto, i. 414; Imray, liii. 80, 1; Madrid, p. 6; Rufz, p. 15; Pugnet, p. 357; Fontana, p. 73; Bruce, p. 278; Blane, p. 415; Gillespie, p. 43; McArthur, p. 348; Rochoux, p. 316; Bally, p. 233; Dickinson, p. 132; J. Clark, p. 16; Catel, Ann. Marit., 1844, iv. 225; Dupont, p. 19; Lallemant, p. 89; Finlay, p. 16; Joubert, p. 968; Jolivet, p. 11; Dyott, pp. 1003, 5.

² Pariset, p. 430; Louis, 195; Baxter, xxi. 3; Drysdale, i. 130; Audouard, p. 64; Gillkrest, ii. 271; Imray, liii. 11; Caillot, p. 19; Gillespie, p. 43; Bancroft, p. 33; Rochoux, pp. 316, 317, 504.

³ R. Jackson (Spain), p. 73.

⁴ Townsend, pp. 156, 7; Finlay, p. 16; Joubert, p. 968; Jolivet, p. 11; Dupont, p. 19; Lallemant, p. 89.

Carpologia occasionally shows itself and proves annoying.¹

Subsultus Tendinum.—As may be presumed from what precedes, well-marked subsultus tendinum, though not common in the yellow fever, is occasionally met with. In some epidemics it is but little seen. "I was surprised," says Dr. Rush, speaking of 1793, "to observe the last stage of this fever to exhibit so few of the symptoms of the common typhus or chronic fever. Tremors of the limbs and twitchings of the tendons were uncommon. They occurred only in those cases in which there was a disposition to nervous disorders and chiefly in the convalescent stage of the disease (iii. 64). Dr. Chisholm also remarks that "subsultus tendinum is by no means a common symptom in the advanced stage." Some have denied altogether the occurrence of these phenomena;² while by others,³ again, they are represented as occurring more or less frequently.⁴ But whether frequently or otherwise, they have been noticed in most epidemics, and in most localities. They may, like the preceding, be viewed as the effect of a nervous complication—depending, in some measure, on the idiosyncrasy of the patient; and are said to prevail more particularly among hard drinkers (*Leblond*).

Tetanic Symptoms.—To what has been said respecting the phenomena located in the nervous system, or depending on a derangement of the latter, it must be added that chronic spasms, or tetanic symptoms of a more or less general character, have occasionally been noticed in this city, as well as in most other places.⁵

Symptoms, simulating those of hydrophobia and of catalepsy, are occasionally encountered.⁶ Nor is it uncommon to find the patient suddenly carried off by an apoplectic condition of the brain. The disease, indeed, as we have seen, assumes, in the highest grade of the congestive form, the apoplectic character.⁷

In closing these remarks relative to the effects of the disease as they manifest themselves in the nervous system, it may be proper to call attention to that which it exercises on the memory. Many individuals experience a partial or

¹ Catel, Ann. Mar., 1844, p. 225; Holliday, p. 10; Savarésy, p. 284; Chisholm, i. 163; Ib., Manual, p. 179; Joubert, p. 968.

² Pariset, p. 430; J. Clark, p. 16.

³ Palloni, p. 5; Lafuente, in Rochoux, p. 505.

⁴ Lining, ii. 425; Addoms, p. 11; Deveze, p. 26; Valentin, p. 169; Kelly, xiv. 378; N. O. 1839, p. 334; Caillot, p. 19; Dyott, p. 1003; Dickinson, p. 136; Bally, p. 233; A. Hosack, p. 15; Bruce, p. 278; Holliday, p. 10; Rufz, p. 15; Moseley, p. 438; Hillary, p. 150; Madrid, p. 27; Savarésy, p. 277; Fontana, p. 73; Gros, p. 11; Barton, p. 39; Perlee, iii. 11; C. Drake, xxi. 135; Thomas, p. 85; Girardin, p. 56; Tully, p. 299; Audouard, p. 63; Proudfoot, xxvii. 250; Berthe, p. 87; Caisergues, p. 169; Leblond, pp. 104, 119; Catel, Ann. Mar., 1844, iv. 225; Mabit, p. 10; Dupont, p. 19; Anderson, p. 8; Lallemant, p. 89; Wragg, x. 81.

⁵ Rush, iii. 210; Rochoux, p. 505; Gillkrest, p. 271; Audouard, p. 64; Hunter, p. 80; Frost, xiii. 151; Chisholm, i. 180; Bally, pp. 167, 174, 233; Med. Reposit., ii. 214; Lallemant, p. 89.

⁶ Catel, p. 16; Bally, pp. 165, 7, 233; Palloni, p. 9; Jolivet, p. 9; N. Y. Med. Rep., ii. 214; Lowber, Med. Mus., v. 22; Kéraudren, pp. 7, 8.

⁷ Bally, p. 233; Wilson, p. 11; Rush, iii. p. 63.

complete, though not permanent loss of that faculty after recovering. Dr. Rush states that several of his patients retained no remembrance of anything that passed in their sickness, although the operations of their understanding had not been impaired throughout the whole course of the fever. In illustration of this, he remarks: "My pupil, Mr. Fisher, furnished a remarkable example of this correctness of understanding, with a suspension of memory. He neither said nor did anything, during his illness, that indicated the least derangement of mind, and yet he recollected nothing that passed in his room, except my visits to him. His memory awakened upon my taking him by the hand, on the morning of the sixth day of the disease, and congratulating him upon his escape from the grave." Dr. Rush remarks that, "in some, there was a weakness, or total defect of memory, for several weeks after their recovery." "Dr. Woodhouse informed me that he had met with a woman, who, after she had recovered, could not recollect her own name" (iii. 65).

Cases occur in which the muscles are affected with partial or complete paralysis. In the summer of 1854, an instance of the kind came under my observation. The patient suffered considerably, at the outset of the attack, from pain in the limbs; so severely, indeed, that the disease was taken for nothing more than rheumatism. Soon, however, symptoms indicating the presence of yellow fever came on. On the third day the pain subsided, but gave place to paralysis of the lower extremities, which remained immovable to the last. In this case, the sensibility or excitability of the surface was neither increased nor diminished, and the patient retained full possession of his mind till within a few hours of death. On dissection, nothing peculiar was found in the brain or spinal marrow. In some instances the complaint assumes the character of *paralysis agitans*.¹ In others, the loss of power over the muscles is attended with increased and morbid sensibility of the skin. In a case of the kind mentioned by Lallemand, the sensibility was so exquisite that the patient could not bear the least touch, nor even the pressure of the lightest cover. The pain, from this source, was particularly severe on the inner margin of the tibia. The slightest touch of the part elicited violent shrieks. The loss of muscular power was complete. The patient, notwithstanding, recovered. The paralysis disappeared, and with it the exquisite sensibility in question.²

Nervous excitability occasionally falls on other parts. Instances have occurred in which the venereal appetite was morbidly excited, sometimes during the disease, though more frequently during convalescence. Dr. Ashbel Smith, who saw the yellow fever at Galveston during the three epidemics of 1839, 1844, 1847, and appears to have studied it well, states that he observed the spontaneous and genuine venereal appetite in several cases. He saw it in the stage of extreme prostration, and, indeed, of irremediable danger.³ Dr. Wragg tells us of a patient who could not retain a dose of medicine, or a particle of food, and was labouring under a strange form of

¹ Lallemand, *Fev. of Rio Janeiro*, p. 91.

² *Op. citat.*, p. 91.

³ *Transact. of N. Y. Acad. of Med.*, i. 63.

mental hallucination, imagining the most impossible things, when, three days before his death, priapism came on, and persisted to the last hour of his existence (p. 82).¹ Dr. Lewis relates one in which the patient, who was nursed by a number of prostitutes, "in a joyous and mischievous mood," amused himself in *squirting* the thin black vomit at such of the *ladies* as he did not fancy, and, if successful in soiling their clothes, would laugh most immoderately. He expressed a strong desire for sexual intercourse. Dr. Lewis found, on examination, "that this was not a mere whim—if physical signs, at least, were any evidence." One hour after, he expired.

The manifestation in question during convalescence has been noticed in this city. Dr. Rush calls attention to it in his account of the epidemic of 1793. "The convalescence from this disease," he says, "was marked, in some instances, by a sudden revival of the venereal appetite. Several weddings took place in the city between persons who had recovered from the fever. Twelve took place among the convalescents in the hospital at Bush Hill. I wish I could add that the passion of the sexes for each other, among those subjects of public charity, was always gratified only in a lawful way. Delicacy forbids a detail of the scenes of debauchery which were practised near the hospital, in some of the tents which had been appropriated for the reception of the convalescents." Dr. Rush adds that it was not peculiar to this fever to produce the morbid excitability of the venereal appetite. It was produced in a much higher degree by the plague which raged in Messina in the year 1743.² Dr. Deveze, who was physician of the Bush Hill Hospital in 1793, makes mention also of the increased venereal appetite manifested among convalescents in that establishment, and remarks that he had made the same observation in Saint Domingo in individuals recovering from malignant fevers (p. 33). The same propensity in both sexes was observed in 1804, at Carthage (Spain), by Riseuno.³

CHAPTER XVII.

PATHOLOGICAL ANATOMY OF YELLOW FEVER.

THE subject which next calls our attention is the morbid anatomy of the yellow fever. It is one of considerable importance, inasmuch as from a knowledge of the changes discovered in the tissues and organs we may not only hope to arrive at a correct view of the seat and character of those morbid actions from which arise the pathognomonic phenomena of the disease, but also to ascertain the identity or non-identity of the latter with other fevers of kindred character. The subject is the more entitled

¹ See Kéraudren, p. 8, for a reference to three cases of the kind.

² Med. Inq. and Obs., iii. 67.

³ Burnett on Mediterranean Fever, p. 236.

to our regard, because the morbid appearances, reported by some writers as characteristic of the yellow fever of temperate climates, have been declared to differ materially enough from those exhibited in the fever of tropical regions to corroborate their views respecting the non-identity of the latter with the former. Doubtless the inquirer who, relying solely on the statements of Dr. Rochoux in regard to the appearances in question, admits the correctness of what he affirms to be the dissimilar anatomical characters of the fever known under the name of yellow fever in those two regions, and coincides in the deductions he has drawn relative to the nature of the various morbid appearances revealed on dissection, can find but little cause to refuse his assent to the pathological views he has so ably and zealously advocated, and to the distinction which he wishes to establish. For fevers which entail changes in the tissues and organs of so opposite a character, and which depend on pathological states so dissimilar, can scarcely be regarded as identical in nature, as arising from the same cause, and as being governed by the same laws. Whether a more extended survey of the results of the *post-mortem* examinations performed in this city and country, and a comparison of them with similar researches in Europe and in tropical regions, than Mr. Rochoux has thought it proper, or has had the requisite knowledge to make, will justify these conclusions, or whether such a survey will not show that on this, as on every other subject on which that author has sought to indicate a marked difference between those fevers, the same fault has by him been committed—representing as typical of each, changes and phenomena which should be viewed as only of occasional occurrence—dwelling unduly on slight points of dissemblance; and, on the other hand, glossing over, explaining away, or omitting entirely, circumstances of a contrary character, are questions which must be decided by an appeal to facts.

Aware of the importance of acquiring a knowledge of the anatomical characters of the disease, the physicians of this city resorted to *post-mortem* examinations as early as the summer of 1793. During the memorable epidemic of that season, numerous dissections were made by Dr. Physick, conjointly with Dr. Cathrall, and an account of the results was given by them to the public through the medium of a daily newspaper (*Brown's Gazette*), and subsequently inserted by Dr. Rush in his description of the disease (iii. 92). At the same time, numerous examinations were made by Dr. Deveze at the hospital of Bush Hill, and an account of them published the next year in an essay to which too little attention has been paid amid all our discussions relative to the nature and origin of the yellow fever, and to which I shall have frequent occasion to refer.¹ In 1798, Dr. Physick pursued similar inquiries at the City Hospital.² During the epidemic of 1802, Dr. Ffirth,³ and, during that of 1805, Drs. Parrish,⁴ Lowber,⁵ and others, attended diligently to the

¹ Recherches et Observations sur les causes, &c., de la Maladie Endémique que a régné à Philadelphie, &c., en 1793—pp. 77, &c. Philad., 1794

² Rush, iv. 44.

³ A Treatise on Malignant Fever, &c. 1804, and Med. Mus., i. 114.

⁴ Med. Mus., iii. 187, &c.

⁵ Ibid., v. 16, &c.

same subject, as did also Dr. S. Jackson¹ and the physicians of the City Hospital in 1820; and more particularly, and with greater minuteness, the physicians of our public establishments—the Pennsylvania, St. Joseph, and City Hospitals in 1853 and 1854.²

While such has been the zeal displayed, from an early period, in this city, as regards the pursuits in question, the physicians of our other cities have not remained in arrears. The writings of Lining, Moultrie, Mitchell, and other of our early physicians, show that anatomical researches were not overlooked by them, and that, in later times,³ the claims which represent Dr. Physick—to the exclusion, let it be borne in mind, of others equally entitled to a share of any credit that may be bestowed—as being the pioneer in the matter, not only in this city, but in other sections of the country—and, in fact, everywhere—are destitute of foundation. Drs. Rand and Warren, of Boston, published, in 1798,⁴ the records of their dissections during the epidemic of that year in that city; and, subsequently, the writings of A. Hosack, Thomas, Gros, Waring, Lawrence, Townsend, A. Smith, Kelly, Nott, Hayne, Hogg, Harrison, Cartwright, Barrington, and others, abound in useful details on the important subject before us. In Europe, and in tropical regions too, similar researches were early made and have continued to engross attention at almost every return of the disease, and the results obtained laid before the profession with more or less minuteness and accuracy.

It may be premised that here, as elsewhere, eases present themselves—especially when the disease has proved quickly or suddenly fatal, or run a rapid course—in which no organic or textural alteration appreciable to the senses has been discovered on dissection, all the organs and tissues exhibiting, apparently, their normal condition, without thickening, softness, swelling, congestion, redness, or too little of any of them to enable us to refer to them any agency in the effect produced.⁵ But such instances are comparatively rare, and may be, nay, have been observed in other diseases, on the nature of which pathological anatomy has thrown much light. In the majority of cases, some—often considerable—morbid changes are found in one or more parts, internal or external, by which the disease can usually be identified, and which are more or less calculated to lead to a knowledge of its pathology.

A. *Surface*.—With very few exceptions, the surface of the body is more or less discoloured, and generally presents a yellow colour varying from a pale or light to a dark orange or brown tint. In many cases, the skin assumes a greenish or mahogany, or leaden hue, or even becomes purple or black. The former, or lighter shades, are usually encountered in sub-

¹ An Account of the Yellow or Malignant Fever, &c., pp. 77, by S. Jackson.

² Observations on the Pathology of Cases of Yellow Fever admitted into the Pennsylvania Hospital during the summer of 1853, by T. H. Bache, M.D. Am. Journ., July, 1854, p. 121.

³ Caldwell's Autobiography, pp. 259, 260.

⁴ Med. Repos., ii. 249; Currie, pp. 73, 798.

⁵ Arnold, p. 24; Guyon, Réponse à Mr. Lefort, p. 49; Bally, Typhus d'Amérique, p. 202, note.

jects carried off rapidly and by an inflammatory attack; the latter in those whose disease was of a malignant or protracted character. In some cases, a pale yellow line, mingling with the other colours, can be traced from the nose to the pubis. The discoloration is more or less diffused over the surface, occupying only the face or eyes, neck and chest, or the whole body. It is generally deeper on the face and trunk than on the extremities. It increases in depth and extent after death, when it usually manifests itself in those who, during life, were not affected with jaundice.¹ The same appearances which have been noticed not only in this city, but also in other parts of the United States, have, in like manner, been recorded in Europe² and in the West Indies;³ and their occurrence everywhere goes far to prove the identity of the disease in those various latitudes. Everywhere cases occur in which the jaundice colour is but faint, or is totally absent. Everywhere it increases after death, or appears then if it had not existed before; everywhere the skin, at times, assumes the dark mahogany and leaden hues described, and everywhere the discoloration is deeper on the trunk and face than on the extremities. That, in some places, some of the peculiar shades mentioned may have been noticed more frequently than in others, may be true; but, from this, no proof of non-identity can be adduced; inasmuch as the same diversity has been noticed at different seasons in the same place, while at all times during the prevalence of a particular epidemic, other cases, exhibiting the darker appearances, have been noticed.

B. Depending and projecting parts, such as the scrotum, penis, fingers, toes, and ears, which, especially a short period before dissolution, are often very dark and discoloured from the stagnation of blood, become, as do also the back and neck, of a dark purplish hue. These ecchymoses sometimes occupy—in spots of different sizes or shape, round or in stripes—other parts of the body: the forehead, upper portion of the face, as also the trunk and extremities.⁴ The latter depend on the disease, and appear before death. The former are cadaveric effects. (*Pariset*, p. 340.)

c. Besides these ecchymoidal spots, the surface is sometimes more or less covered with others of a minute size, bearing some analogy to petechiæ, or

¹ Rush, iii. 84; Kelly, xiv. 381; S. Jackson, p. 53; Rochoux, p. 349; Deveze, p. 58; Ralph, ii. 79; Lawrence, x. 70, 220; Townsend, p. 192; J. Davy, Ed. Journ., lxxii. 280; Dalmas, p. 14; A. Smith, xxv. 502, 3; Hogg, i. 412; R. Arnold, 317; Bache, xxvii. 121.

² Gillkrest, ii. 274, 5; Louis, p. 53; Fellowes, p. 60; Palloni, p. 10; Burnett, pp. 44, 242; Pariset, p. 340; Rochoux, p. 523; Arejula, p. 419.

³ R. Jackson, Tr., p. 264; Pugnet, p. 361; Vatable, p. 347; Gillespie, p. 69; Ralph, ii. 779; Savarésy, p. 463; Frost, xiii. 32; Dariste, p. 103; Maher, p. 886; Rochoux, p. 349; Fever of Cayenne (1850), p. 161; Copland, iii. 143; Blair, p. 90; Chambolle, xiii. 200; Joubert, pp. 970, 983, 985, 6.

⁴ S. Jackson, p. 53; Kelly, xiv. 381; Deveze, p. 38; Gillkrest, ii. 274; Lawrence, x. 75, 270; Ralph, ii. 79; Bache, xxvii. 221; Valentin, p. 170, 178; Townsend, pp. 151, 190; Rochoux, pp. 349, 524; Maher, p. 886; Louis, p. 54; Harrison, ii. 136; Savarésy, p. 463; Pariset, p. 340; Chambolle, xiii. 200; Catel, p. 12; Burnett, pp. 44, 45, 305, &c.; Rufz, p. 17; Berthe, p. 182; Caisergues, p. 174; Dariste, p. 102; Fellowes, p. 69; Velasquez, pp. 17, 18; Pariset, 46, 50; Fever of Cayenne (1850), p. 162.

partaking in some respects of their nature, and increasing in number after death.¹

The cellular membrane and fat on incision are found, in many cases, to partake of the yellow colour of the skin.² This occurs more frequently in ordinary than in malignant or congestive cases.

D. Extravasations of blood in the cellular membrane under the skin and in the interstices of the muscles, are not uncommonly found both in temperate and tropical regions, and have been noticed in this city.³

E. In some cases, livid and gangrenous spots are found on portions of the body, and in instances where during life the skin was the seat of erysipelas, or other forms of inflammatory changes, of anthrax, &c., traces of these are discovered after death.⁴

F. The joints and muscles are generally rigid and stiff, becoming more so soon after death.⁵

G. The face, in some cases, is found tumefied,⁶ while in others it is, on the contrary, shrunken, both in the yellow fever of tropical climates, and in that of this country and Europe.⁷

H. In individuals who have died of the malignant form of the disease, the muscles are often found of a dusky or dark hue. They are softened in texture and easily torn or broken down by pressure. In other cases, and especially after ordinary and inflammatory cases, those parts exhibit little or no change in point of colour and firmness.⁸ They are sometimes found of a pale colour, and having somewhat the appearance of having been submitted to a prolonged maceration.⁹

Cerebro-Spinal Organs.—The brain, in all its parts, is often found free from diseased changes. Such was the case in this city in 1793.¹⁰ We are also told that in 1805 dissections revealed no unequivocal appearances of inflammation in that organ.¹¹ The same may be said of the results of the examinations instituted in 1820;¹² and similar observations have been recorded in

¹ Rochoux, pp. 349, 524; Fever of Cayenne (1850), p. 162.

² Vatable, p. 349; Gillkrest, ii. 295; S. Jackson, p. 53; Arejula, p. 419; Dariste, p. 103; Fellowes, p. 69; O'Halloran, p. 187; Deveze, p. 59; Townsend, p. 190; Gillespie, p. 69.

³ Fellowes, p. 57; Gillkrest, p. 277; J. Smith, xxxv. 45; Rochoux, p. 330; Maher, p. 886; Furlong, p. 290; R. Hosack, p. 16; Catel, p. 12; Pariset, p. 341; Deveze, p. 80; Valentin, p. 170; Fever of Cayenne, p. 162.

⁴ Rochoux, p. 524; Maher, p. 886; Pugnet, p. 361; Chisholm, i. 169; Gillespie, p. 153; Desportes, i. 200, 214, 223; Hillary, p. 152; Keraudren (Arch. Gén.), xv. 459; Savarésy, p. 277, &c. See Chapter on Affections of the Skin.

⁵ Deveze, p. 59; Rush, iii. 89; Louis, p. 53; Palloni, p. 10; Nott, ix. 281; Rochoux, p. 350; Savarésy, p. 463; Bache, xxvii. 121.

⁶ Rochoux, p. 349.

⁷ Ibid., p. 524; Savarésy, p. 463; Pariset, pp. 17, 46, 50.

⁸ Gillkrest, ii. 274, 5; Kelly, xiv. 381; Copland, iii. 143.

⁹ Fever of Cayenne (1850), p. 162.

¹⁰ Rush, iii. 92.

¹¹ Caldwell, p. 98; Lowber, Mus., v. 21; Parrish, Mus., iii. 188.

¹² S. Jackson, p. 78.

other parts of the United States, in Europe, and tropical climates,¹ where it is not unusual for the brain to present no appreciable lesions; or, at any rate, none indicative of the existence of prior inflammation. On the other hand, in a few cases in 1798, in 1802, and at other seasons here, and more or less frequently in other places, the brain and its membranes have presented marks of acknowledged inflammation—great redness of the substance, partial injection, thickening, opacity, grayish spots, together with engorgement of the vessels and sinuses, effusion, &c.²—or appearances which, though not so specifically stated, we may infer to have been of that character.³

But, though traces of inflammation are thus found to present themselves; though by some writers they are represented as occurring in all cases, and constituting one of the main anatomical characters of the disease, they are acknowledged by many more, and, so far as my own observations go, very justly, as being absent in a greater number of cases—perhaps in the larger number—the proportion varying in different epidemics. Dr. Evans states that he discovered meningitis in 17 out of 37 cases he dissected, and that inflammation of the cerebral mass itself was of rare occurrence (p. 230). Dr. McArthur found inflammation to fail in half the number of dissections even among those who had had delirium (p. 349).

Dr. Maher, who, in Martinique in 1827, found in almost every case he dissected—30—marks of arachnoiditis, and not unfrequently softening of the encephalic mass, admits that subsequently, at the Havana, he discovered similar alterations in two cases only (p. 885). It is remarked by Dr. Gillkrest,⁴ that in the course of the epidemic at Gibraltar, in 1828, as well as on “other occasions in the West Indies and elsewhere, extensive observations carefully conducted have quite negatived any assertion made from time to time as to morbid changes in the substances of the brain; as an inordinate quantity of fluid in its cavities, or under its coverings; remarkable congestions; extravasation of blood; the effusion of lymph, &c.; even where profound coma had taken place in the Gibraltar cases, morbid states by which this might be explained, were not discovered; and the deviations from perfectly natural states, observed in any cases, were considered by those who had most opportunities of making the examinations as nothing more than the fortuitous appearances which present themselves in a proportion of instances, no matter from what disease death is produced, and which, as is now generally admitted, may arise from stasis, or the longer duration of the last agonies in particular instances.” “Cadaveric changes, too,” Dr. G. adds,

¹ Davy, Ed. Journ., lxxii. 280; Waring, p. 48; Pariset, p. 343; Rochoux, pp. 525, 6; Bally, pp. 187, 8; Maher, p. 885; Gillkrest, ii. 276; Harrison, ii. 137.

² Rush, iv. 44; Ffirth, p. 35; Dickson, p. 357; Burnett, pp. 45, 306; Evans, p. 230; Louis, p. 306; Bancroft, p. 36; Maher, p. 885; Catel, p. 12; Rochoux, p. 370; Palloni, p. 17; Gillespie, p. 70; Ralph, ii. 79; Vatable, p. 347; Imray, liii. 91; O'Halloran, p. 193; Cartwright, ix. 39; Osgood, p. 12; McArthur, p. 349; Chambolle, xiii. 200; Lefort, de la S., p. 577; Desportes, i. 204; Frost, xiii. 252; R. Jackson, Sketch, i. 89, 106, 113.

³ Dalmas, p. 16; Savarésy, pp. 469, 470.

⁴ Cyclopedia, ii. 276.

“have, no doubt, given rise to mistakes, particularly to too great vascularity or congestion in the posterior and more dependent parts of the membranes of the brain, as it has regarding the most dependent folds of the intestines.”

From these facts, and from the circumstance that in the dissections which I noticed, and in others performed in this city and various parts of this country and elsewhere,¹ traces of inflammation in the brain, and in its enveloping membranes, were seldom if ever found; that, by those who speak of them as common occurrences, it is admitted that in a greater or less number of cases such traces are not discovered, and that the opinion of the existence of cerebral and meningeal inflammation has often been predicated on the discovery of changes which were probably due to other morbid conditions, we may safely conclude that inflammation of those parts, though doubtless occurring sometimes in yellow fever, and leaving its usual textural effects behind, is far from being a frequent attendant on the disease, and must, therefore, not be viewed as characteristic of the latter. But, while traces of acute inflammation of the cerebral organs are but seldom found, it is not unusual to discover in those parts changes of an abnormal character, and which have often been mistaken for evidences of true phlegmasia. In many cases, the perieranium, the sinuses, and vessels of the brain are found more or less gorged, or congested with blood.² In a certain number the membranes, alone or together with the brain, are injected throughout or in patches;³ and, in not a few, water of a limpid or yellow colour, or mixed with blood, is effused in the ventricles, at the basis, or on the surfaces, or in the membranes.⁴

Spinal Marrow.—In the dissections performed in this city, the spinal marrow was sometimes found more or less gorged with blood, and its membranes apparently the seat of what some would denominate inflammation. Dr. Cartwright, of Natchez, examined that organ in four cases, and in all the arachnoid and pia mater presented, as he informs us, the usual appearances of inflammation, though in a less degree than the same membranes within the cranium. The lumbar and sacral portions appeared more diseased than the cervical and dorsal. The delicate tissues investing the nerves going off from the medulla spinalis where those nerves passed through any inflamed

¹ Joubert, pp. 985, 6; Lowber, *Med. Mus.*, v. 31, 32; R. Arnold, p. 318; Bache, xxvii. 121, &c.; Cayenne (1850), p. 165.

² Blair, p. 99; Hacket, *Med.-Chir. Rev.*, xvi. 290; Harrison, ii. 137; Pariset, p. 343; Dalmas, p. 16; Rochoux, p. 370; Pugnet, pp. 361, 2; Ralph, ii. 79; Bancroft, p. 36; Fever of Cayenne (1850), p. 165; Rufz, *Examiner*, iii. 70; Catel, p. 12; J. Davy, *Ed. Journ.*, lxxii. 280; Lawrence, x. 3, 5, 255; Imray, lxiv. 330; Bache, xxvii. 121, 4.

³ Bally, p. 187; Burnett, pp. 45, 306; Kelly, xiv. 381; Harrison, ii. 137; Pugnet, pp. 361, 2; Bancroft, p. 36; Rufz, *Examiner*, iii. 70; Cartwright, p. 39; Lawrence, x. 255.

⁴ Bally, p. 187; Maher, p. 885; Harrison, ii. 137; Bache, xxvii. 124; Dalmas, p. 16; Pugnet, p. 361; Bancroft, p. 36; Rufz, *Examiner*, iii. 70; Catel, p. 12; Waring, p. 48; Pariset, p. 343; Cartwright, ix. 39; Lawrence, x. 7; Thomas, p. 136; Rochoux, pp. 372, 526, 527; Cayenne (1850), 165; Hacket, xvi. 290; Blair, p. 100.

part, were found of a scarlet colour.¹ The results obtained in New Orleans by Thomas were not very different—effusion of blood in the vertebral canal, redness of the arachnoid and other membranes—sometimes more particularly in the dorsal region, and extending along the nerves (pp. 131–6). Those noted by O'Halloran in Spain do not differ very materially from the foregoing (pp. 187, 195). Other physicians have found a sanguine effusion in some portion of the canal, and an injection of the membranes or of the whole organ, and generally a greater or less quantity of serous fluid—rarely a change of consistence in the marrow itself;² while others, again, have not found the parts to vary from the natural standard, or to present appearances different from those exhibited after other acute diseases.³ In only one case among those recorded by Louis (p. 306), the dura mater was of a clear and livid red throughout its whole extent. There was about a spoonful of serum of a pale red colour in the lower part of the arachnoid, and the spinal marrow was flaccid through its whole extent, without having lost its cohesion.

Whether the changes found in the spinal organs are to be referred to inflammation, may very properly be doubted. More properly are they to be referred to mere congestion, which has but too often been mistaken for the former, and to the hemorrhagic tendency which, as we have seen, constitutes a main characteristic of the disease. If, indeed, the medulla, or its membranes, have presented unequivocal marks of the inflammatory process, such cases may reasonably be viewed as exceptional—the effect of an accidental complication—and not as forming an essential part of the disease. Nor is it less true, that the unaltered condition of the parts in most, or at least very many, cases; the fact that their redness, in some instances, is no indication of the prior existence of acute morbid action, and is often found after other and very dissimilar diseases; added to the circumstance that the serous effusion described as discovered in many instances cannot be viewed as a proof of prior disease, since it is found in subjects who have died from the most opposite causes, lead to the conclusion, that so far as our information extends, we are not to look to the spinal organs for the seat of any morbid change of importance, or peculiar to, or characteristic of, the yellow fever.

Ganglionic System.—Dr. Cartwright, of Natchez, found the ganglia and the ganglionic nerves in a diseased state, and their investing membranes inflamed. “The semilunar ganglions and celiac plexus were, in particular, highly diseased. The membranes immediately investing these ganglions and their plexuses, were of a deep scarlet, and, in some places, of a black colour. This inflammation was not confined to the tissues immediately investing the nerves, but extended to the neighbouring tissues, especially of the semilunar ganglion. The whole of the membranes inclosing the nerves denominated the solar plexus, lying upon the celiac and superior mesenteric arteries, were black with inflammation. The cellular substance

¹ Med. Recorder, ix. 40.

² Pariset, pp. 343–5; Bally, p. 187; Harrison, ii. 137; Cayenne, 1850, p. 166; Blair, p. 100.

³ Cyclopaedia, ii. 277; Louis, p. 63; Rochoux, p. 525.

investing the hepatic plexus, as it extends over the hepatic artery and vena portarum, the splenic, the mesenteric, and renal plexuses, together with the cardiac and pulmonary plexuses, were found to be of a scarlet colour. In a word, the delicate tissues investing the whole of the ganglionic system of nerves were more or less inflamed." This inflammation was found in all of seventeen subjects examined.¹ The editor of the *London Medical Repository* (June, 1825), also informs us that he has often found the ganglia inflamed on dissecting individuals who had died of yellow and other fevers.

From the uniform integrity of the cerebral functions in the first stages of the yellow fever—the extremely frequent integrity of those functions to almost the last moments of existence in its "congestive" or most intense and fearful form—together with the remarkable manner in which (in the last-mentioned form especially) the secretions are suspended, Dr. Gillkrest was induced to infer that the ganglionic system was involved very prominently in the series of morbid actions.² But he does not appear to have found any pathological changes in those parts calculated to render his opinion otherwise than conjectural. Other inquirers have found no appreciable lesions in the ganglia;³ and, in none of the examinations that I have witnessed, was anything of importance found in these, or in the plexuses and nerves. From this, and from the fact that the same parts have been found inflamed, and otherwise diseased, in other maladies having not the least resemblance to the yellow fever—as tetanus, for example,⁴ and diarrhœa, as also in fevers but remotely allied to the latter⁵—and considering, besides, that it would seem to have become somewhat of a fashion to place in the sympathetic ganglia all diseases the pathology of which is obscure, we may conclude, not only that the morbid changes in question are not always present in yellow fever, but also that they exist without giving rise to the disease, and hence, when present, are not to be viewed as among its true anatomical characters.

THORACIC ORGANS. Lungs.—The dissections made in this city go to show that, in general, the respiratory organs present no appearances indicating that they partake largely and necessarily in the diseased action of the system in yellow fever. In 1793, they were found perfectly sound by Drs. Physiek and Cathrall.⁶ A similar exemption in the lungs from disease of importance has been noticed by other authors in various parts of the country, in Europe, and the West Indies.⁷ In other cases, the substance of these organs is in a state of inflammation, presenting the first, and, in a few cases, the second degree of pneumonia.⁸ Much more generally the lungs, especially at the poste-

¹ Med. Recorder, ix. 37.

² Cyclopædia, ii. 278.

³ Harrison, ii. 137.

⁴ Swan, An Essay on Tetanus, founded on cases and experiments. London, 1825.

⁵ See Campbell, Trans. of Am. Med. Assoc., vi. 472, &c.; Lobstein.

⁶ Rush, iii. 92; Deveze, p. 60; S. Jackson, p. 78; Caldwell, p. 99; Bache, p. 121; Lowber, v. 20, &c.

⁷ Harrison, ii. 137; Rochoux, pp. 367, 368; Pariset, p. 347; Gillkrest, ii. 276; Chambolle, xiii. 201; Joubert, pp. 984-6.

⁸ Louis, p. 65; Rochoux, pp. 368, 528; Palloni, p. 8; Periodico de la Soc. Chir. di Cadiz, ii. 261; Dickson, i. 357; Savarésy, p. 460; Lefort, p. 577; Frost, xiii. 252; Bancroft, p. 33; Cartwright, ix. 40.

rior or lower portion, are found more or less gorged with dark-coloured and altered blood.¹ Dr. R. Jackson's remarks on the subject of the gangrenous form of West Indian fever will apply to a state of things not unfrequently noticed here and elsewhere. "The lungs were frequently black, resembling a sponge—sometimes throughout, sometimes partially; in substance sometimes firm and dense, not unlike the substance of the spleen (i. 89), or gorged with blood, black and dissolved" (i. 94). When thus engorged, they do not retreat or collapse when the sternum is removed (*Harrison*, 137). Not unfrequently the surface of the lungs is covered with black melænic patches or ecchymoses of from two to five lines in diameter, or masses of the same colour impermeable to the air.²

The mucous membrane of the air passages, though sometimes injected, or spotted with blood, or even inflamed,³ is in general free from any morbid change. In Gibraltar, in 1828, according to Louis (p. 64), this preservation of the normal condition was more uniform and remarkable than is found to be the case in individuals who die of the acute febrile diseases of Paris.

The pleura is usually unaltered,⁴ the ecchymoid spots noticed upon it being situated in the cellular membrane beneath or in the substance of the lungs, and not affecting the tissue. In some cases, however, it has been found inflamed, or containing a greater or less quantity of effused serum of a yellowish, orange, or reddish colour,⁵ with or without marks of inflammation. In a few, again, the fluid effused is of a sanguinolent character, and resembles, more or less closely, the black vomit.⁶

Heart.—The substance of the heart, like that of other muscles, is sometimes of a dusky colour, and softer, and more flabby than natural, and easily broken down by pressure between the fingers.⁷ In other, and perhaps a greater number of instances, the organ retains its natural appearance.⁸

Professor Riddell, during the epidemic which prevailed in New Orleans in 1853, examined the substance of the heart under the microscope, and found the muscular fibres affected with molecular degeneration or disarrangement—all traces of striation having disappeared, and giving way to a granular condition.

¹ Palloni, p. 10; Rufz, p. 17; Bally, p. 190; Deveze, p. 60; Harrison, ii. 137; Vatable, p. 348; Gillespie, p. 70; Rochoux, p. 368; Pariset, p. 348, 368; Ffirth, p. 35; J. Davy, Ed. J., lxxii. 280; Ralph, ii. 80; Fever of Cayenne (1850), p. 163; Blair, p. 99; R. Arnold, p. 318; Hacket, xvi. 290; McArthur, p. 349; Savarésy, p. 460; Lefort, p. 577; Caisergues, p. 182; Dalmas, p. 16.

² Arejula, p. 427; Louis, p. 65; Rochoux, pp. 368, 528; Bally, p. 190; Gillkrest, ii. 276; T. Smith, Ed. J., xxxv. 44; Cayenne (1850), p. 163; Hayne, Charleston J., vii. 18; Fellowes, p. 68; Deveze, p. 60.

³ Harrison, ii. 137; Pugnet, p. 362; Savarésy, p. 460; Blair, p. 99; Smith, Ed. J., xxxv. 44; Cartwright, ix. 40; Bache, xxvii. 122, 3, 6.

⁴ Louis, p. 73; Rochoux, p. 367, 528.

⁵ Palloni, p. 10; Pugnet, p. 362; Bache, xxvii. 122; Bally, 190; Rochoux, p. 528.

⁶ Parrish, Med. Mus., iii. 191; Merrill, Med. Essays, p. 7.

⁷ Gillkrest, ii. 274; Rufz, p. 17; Louis, p. 75; Evans, p. 228; Ralph, ii. 80; Rochoux, p. 369; Blair, p. 99; Copland, iii. 143.

⁸ Palloni, p. 11; Harrison, ii. 137; Evans, p. 228; Rochoux, pp. 369, 529.

“I first observed this remarkable change of the molecular structure of the heart in yellow fever on the 11th of August, 1854, in the heart of Maria Mier (æt. twenty-five), a section of which, twelve hours after death, was brought to me by Dr. Maegibbon. Mostly through the autopsies made by the same gentleman, I have since had opportunities to examine nearly thirty yellow fever hearts. In general, the molecular change is either complete or well-marked; rarely, it is very slight or imperceptible. Different portions of voluntary muscle from the same cases manifested no perceptible change, no loss of striation.” The molecular degeneration of the heart can always, according to Prof. Riddell, be satisfactorily made out, affirmatively or negatively, with a good microscope.¹

The pericardium, which generally appears healthy, sometimes contains a notable, though not unusual quantity of serous fluid of a yellow or reddish colour.² In one case, mentioned by Catel (p. 12), the membrane was inflamed and partly filled with blood, red and coagulated. In others, there is an effusion without inflammation (*Pariset*, p. 348).

In a case described by Dr. Bache, and which I saw, “the pericardium contained four fluidounces of a very turbid greenish brown fluid, resembling black vomit, which, being poured into a bottle, and allowed to stand a short time, separated into two portions, that at the bottom of the bottle of a white or pale yellow, while the rest remained of the original colour. An examination by the microscope proved the brownish fluid to be altered blood-corpuscles, with less of the granular amorphous matter than is usually found in the genuine black vomit.” No epithelial scales could be detected. “The whitish fluid was pus. The heart itself was stained of a dark colour at its base, and the pericardium was minutely injected in points” (*Bache*, xxvii. 123).

The endocardium, in some cases, is slightly red, apparently the effect of staining. In many others it is, as well as the fibrous parts, the valves, &c., of a yellow colour, which often extends into the aorta and the larger vessels. On the surface of the former, spots, bearing a strong analogy to petechiæ, are sometimes discovered.³ The cavities of the heart contain a greater or less quantity of blood, which, except in a few epidemics, or in some cases during every season, has usually been found dark, and, for the most part, grumous or fluid, with or without coagula of the same colour.⁴ In a large number of cases these cavities—the ventricles particularly—contain albuminous concretions, varying in size and consistence, and of a transparent yellow colour, having the appearance of meat-jelly or fine amber.⁵ They penetrate some-

¹ Riddell, Microscopical Obs. pertaining to Y. F.

² Makittrick, p. 95; Arejula, pp. 427, 430; Palloni, p. 11; Pugnet, p. 362; Ralph, ii. 80; Frost, xiii. 253; Bally (p. 190), Savarésy, p. 460; Rochoux, pp. 369, 524; Ffirth, p. 35; Cayenne (Fev. of 1850), p. 162.

³ Harrison, p. 137; Fey. of Cayenne (1850), p. 162.

⁴ Pariset, p. 350; Rufz, p. 17; Ffirth, p. 35; Arejula, pp. 426–8; Pugnet, p. 362; Vatable, p. 348; Ralph, ii. 80; Louis, p. 75; Blair, p. 99; Bally, p. 193; Rush, iii. 92; Copland, iii. 144; Frost, xiii. 253; Hacket, xvi. 290; Stevens, p. 354; Townsend, p. 190; Cartwright, ix. 43; see *ante*, chapter on the *Blood*, p. 163.

⁵ Rochoux, p. 529; Gillkrest, ii. 276; Louis, p. 75; Gillespie, p. 71; Vatable, p. 348;

times into the aorta. Bally dwells with emphasis on these concretions (p. 193), the existence of which, he thinks, he was the first to discover. Dr. Pennell, to whose essay reference has already been made, states that he became attached to the hospital at Rio Janeiro in February, 1852, and that between that month and the end of May, he made upwards of fifty-four *post-mortem* examinations of individuals who had died of yellow fever, and whose disease he had watched during life. "A clot was found in every instance, with the exception of those cases which had been largely blooded, and even these did not always form an exception." The clot was generally more or less round, of a clear bright amber colour, of a gelatinous appearance, but, upon closer examination, was found to be much tougher, and evidently fibrinous. Sometimes the extremities of the clot had small portions of coagulated blood adhering to them. When found in the heart, the clot, according to Dr. P., always exists in the right ventricle, or ventricle and auricle, but is usually accompanied by another of smaller size in the left. There may also be clots in the *venæ cavæ*, and sometimes, but less commonly, in these alone.¹

ABDOMINAL ORGANS. Stomach.—Of all the organs of the body, the stomach may justly be regarded as the one most generally and seriously implicated in the yellow fever, and in which the marks of disease are most frequently discovered after death. Externally, it is sometimes found of a yellow colour; but in other cases—and, indeed, generally—it retains its normal appearance. On opening the organ, it is almost uniformly found to contain a greater or less quantity of matter similar to that thrown up during the latter stage of the disease, and known under the name of black vomit.² In some cases, the contents of the stomach consist of blood more or less pure, with or without coagula, and generally combined with a portion of glairy matter, and substances swallowed a short time before death. In some instances, the mucous coat is smeared over with a dark, adhesive, jelly-like substance containing portions of blood.³ Under this substance, and sometimes when it does not exist, we find a layer of grayish matter not unlike a mixture of linseed meal.⁴ In some cases, again, the lining membrane, when cleansed from these various coatings, is found to present its normal appearance, and to be perfectly free from any appreciable morbid lesion. Indeed, in not a few instances, it is even whiter than in its normal state.⁵

J. Davy, *Edinb. J.*, lxxii. 280; Ralph, ii. 80; R. Jackson, *Sketch*, p. 94, 1st ed.; Catel, p. 12; Doughty, pp. 118, 132, 145; Ruz, p. 17; Pariset, p. 349; Ffirth, p. 35; Copland, iii. 144; *Fever of Cayenne* (1850), p. 162; Cartwright, ix. 43.

¹ *Med.-Chir. Trans.*, xxxvi. 247. See *Lancet*, April, 1853, Am. ed.

² See *ante*, chapter on Black Vomit.

³ Gillkrest, ii. 275; Thomas, p. 132; Cartwright, ix. 42; Rush, pp. 90, 92; Hayne, vi. 346, &c.; Blair, p. 101; Chambolle, xiii. 201; Rochoux, p. 352; *Physick, Reposit.*, v. 131; Townsend, p. 190.

⁴ Pariset, p. 353; Rochoux, pp. 352, 532; Chambolle, xiii. 201.

⁵ Harrison, ii. 139; Louis, p. 96; Waring, p. 49; Nott, ix. 280; Gillkrest, ii. 275; Savarésy, p. 464; R. Jackson, pp. 94–5; Frost, xiii. 253; Desportes, i. 202; *Physick*,

The integrity of the mucons membrane in such cases—the absence of capillary congestion or inflammation—has been ascribed to the effusion of the matter of black vomit or of blood, a quantity of which is nearly always found in the viscus.¹ But this opinion, which would seem to receive support from the known origin of that matter, and from the fact that the quantity is often less in cases where the membrane is found most diseased, is supposed to be negatived by the fact that, in some cases, the quantity is just as large when the membrane is diseased as when healthy, and that in some, in which the stomach was filled with black fluid, the vessels of the mucous membrane were found literally gorged with blood. Such instances, however, are, after all, seldom encountered, and the simultaneous existence of capillary congestion in the mucous membrane and sanguineous effusion in the cavity of the stomach is no proof that the latter does not usually relieve the former, but merely shows that, in cases in which it occurs, the hemorrhagic tendency has been so strong that the blood effused from the engorged capillaries has been rapidly replaced.

The number of cases in which the membrane is exempt from appreciable morbid change is generally limited, and varies in different places and seasons. According to Dr. Harrison,² such cases are seldom met with at New Orleans (p. 139). Louis found three only in the whole number he dissected (p. 96). Dr. Blair mentions only four (p. 92). Dr. Nott, on the other hand, found four cases out of eight at Mobile in 1843, and three out of eight in 1844 (pp. 279, 80). They are encountered more particularly in malignant cases in which arterial reaction is but feeble, or in rapid cases of other varieties.³

Much more frequently, the mucous membrane is found more or less diseased, and presenting changes, which, though regarded by some as the product in most, if not all cases, of capillary congestion, of mere staining, or, indeed, of any other morbid process than inflammation,⁴ are admitted by a large number of pathologists as indicating—not all, but some—the prior existence of the latter diseased condition. Hence, by most writers on the yellow fever, the stomach is stated in positive terms to present after death decided marks of inflammation; or alterations are described which are known to be the effects of that process.⁵

v. 131; Ticknor, iii. 229; Lowber, Mus., v. 19, 23; Ffirth, p. 35; Parrish, Mus., ii. 188; S. Jackson, p. 78; Deveze, p. 87; Cartwright, ix. 412; Copland, iii. 143; Blair, p. 92; Rochoux, p. 355; Dict. des Sci. Méd., xv. 341; Hume, p. 210.

¹ See *ante*, chapter on Black Vomit, pp. 304–5.

² Harrison, p. 139; Hayne, Charleston Journ., vi. 628; Gillkrest.

³ Cyclop., ii. 275.

⁴ Daniel, pp. 84, 5; Rufz, p. 18; Harrison, p. 138; Lempriere, ii. 104; Keraudren, p. 7; Gillkrest, ii. 275.

⁵ Imray, liii. 90; Desportes, i. 102; Moseley, pp. 441, 2; J. Hunter, pp. 160, 1; Bancroft, p. 39; Jackson, Tr., p. 265; Gillespie, p. 69; Vatable, p. 348; Osgood, p. 12; Ralph, ii. 80; McArthur, p. 349; Lefort, p. 577; Jackson, Sketch, p. 62; Frost, xiii. 253; Savarésy, p. 461; Dubreuil, viii. 329; Barrington, xii. 316; Waring, p. 49; Catel, p. 12; Evans, p. 216; Louis, p. 97; Bally, pp. 193, 4; J. Mitchell, Additional Obs.,

That some of these changes are of a nature to justify the opinion of those who deny the truly inflammatory character of the local disease in all cases, there can be no doubt. Nor is it improbable that many of those who have assumed that the changes found are due exclusively to inflammation, have predicated that opinion on appearances which more ample experience would have shown to be the result of a different morbid process. But it is not less true that, in a large number of individuals who have died of the yellow fever, the lining membrane of the stomach bears the most unequivocal marks of inflammation. On both these points, the following enumeration of the morbid changes found in that organ will leave no doubt:—

A. In some cases, the stomach is found contracted;¹ in others it is distended.²

B. The longitudinal rugæ are enlarged. The surface has often a vermicular appearance, being corrugated and thrown into numerous folds.³

C. The capillary vessels are to a greater or less extent injected with blood. Sometimes these vessels are literally gorged; to such a degree, indeed, that portions of the membrane “cut and dried, have formed very perfect preparations, exhibiting the ramifications of the vessels into their minutest divisions in a very beautiful manner” (*S. Jackson*, p. 21).⁴

D. The redness is, in some cases, of a very intense and dark hue, and has been compared to the redness produced in that organ by the ingestion of acrid poisons.⁵ In other instances, the colour is lighter; assuming a rose, claret-brown, ruddy, pale-ash, greenish or yellowish hue. In other instances, again, the colour is leaden, livid, or even nearly black.⁶

E. This discoloration is either uniform in appearance or in the form of

p. 384; Warren, *Review of the Mercurial Practice in Feb. Dis.*, p. 115; Pariset, p. 352; Belcher, xxiii. 252; Palloni, p. 11; Arejula, pp. 426, 9; Riseuno, in Burnett, pp. 242, 3; Caldwell, p. 99; Rush, iii. 12, 90; *Ib.*, iv. 44; Deveze, p. 63; Ffirth, p. 36; Lowber, *Mus.*, v. 19; Steward, *Mus.*, ii. 303; *S. Jackson*, p. 78; Lawrence, x. 37, 253, &c.; Physick, *Repos.*, v. 130; Cartwright, ix. 41; Thomas, p. 132; Dalmas, p. 15; Townsend, p. 190; Barton, xv. 39, 40; Parrish, *Mus.*, iii. 188; Smith, xxv. 504; Ticknor, iii. 231, 2; O'Halloran, p. 186; Pym, *Ed. J.*, xxxv. 12; T. Smith, *ib.*, xxxv. 44; Proudfoot, xxvii. 257; Moultrie, p. 14; Imray, liii. 90; *Ib.*, lxiv. 330; Pugnet, p. 363; Ramsey, *Charleston Journ.*, ii. 627; R. Arnold, pp. 317, 318; Chambolle, xiii. 201; *Fever of Cayenne* (1850), p. 163; Dickson, p. 357; Bache, xxvii. 123.

¹ Caldwell, *Fev. of 1805*, p. 89; *Med. Mus.*, ii. 303; Deveze, p. 63; Lawrence, x.; Rush, iii. 93; Rochoux, p. 351; Hacket, xvi. 290; Blair, 92.

² *Museum*, v. 20; Lawrence, x. 7; Rochoux, p. 351; Ralph, ii. 80; Hacket, xvi. 290; Blair, p. 92; Hayne, vi. 638; Bache, xxvii. 121.

³ *Museum*, ii. 303; *Ib.*, v. 19; Lawrence, x. 9; Imray, liii. 90; *Fever of Cayenne*, p. 163; Bache, xxvii. 121.

⁴ Deveze, p. 63; Ffirth, p. 36; Rush, iii. 92; *Museum*, i. 117; *Ib.*, iii. 188; *Ib.*, v. 19, 20, 23; Lawrence, x. 6, 259; Kelly, xiv. 382; Ralph, ii. 80; Rochoux, p. 531; J. Davy, *Edin. Journ.*, lxxii. 280; Furlong, p. 290; Cartwright, ix. 41; Physick, *Med. Repos.*, v. 131; Blair, pp. 92, 93, 101; Pinkard, ii. 226; R. Arnold, pp. 317, 18; Nott, *Am. Journ.*, N. S., ix. 279; Bache, xxvii. 121; Hayne, *Charleston Journ.*, vi. 342, &c.

⁵ Physick, in Rush, iii. 92.

⁶ Rochoux, pp. 353–54, 531; Blair, p. 101.

arborizations;¹ extending over a large portion or the whole of the membrane, or, as oftener takes place, it is limited to the cardiac or pyloric orifices, and to the larger curvature, and presents itself in patches of greater or less size, and more or less numerous. In addition, whatever may be the tint of the membrane, we discover streaks or spots of a purple colour, spreading in various directions over both the altered and healthy parts.² These spots assume frequently the appearance and characteristics of true ecchymosis—varying in size and number. At other times, with or without those large patches, are found numerous small, dark red, or violet round spots,³ superadded to the rose hue of the surface, with which it forms a distinct contrast. They resemble flea-bites, and have the same appearance as the petechial spots noticed on the external surface of the body.

F. The membrane is the seat, besides, of abrasions or small depressions, or pits, like holes or furrows, which convey the idea of a portion of the tissue having been removed.⁴

G. It very frequently is found mammillated even to a remarkable degree. Louis found it so in two-thirds of his cases.⁵

H. The membrane, in such cases, or even when no mammillated elevations exist, is found more or less considerably thickened and opaque.⁶

I. In some of the latter cases, or even when not thickened, the membrane is softened, and easily detached, especially about the great *cul-de-sac*.⁷ In a few cases, it is the seat of ulceration,⁸ or of gangrenous changes.⁹

K. In some instances, more particularly when the disease was of a malig-

¹ Maher, p. 884.

² Lawrence, ix. 4, 253, 258; Rush, iii. 92; Dalmas, p. 15; Imray, liii. 90; Bache, xxvii. 123; Maher, p. 884; Vatable, p. 348; Bally, pp. 193–94; Evans, p. 316; Pariset, p. 352; Rufz, p. 17; Rochoux, p. 354; J. Davy, lxxii. 280; Fever of Cayenne (1850), p. 163; Blair, p. 101.

³ Blair, p. 101; Pariset, p. 352.

⁴ Med. Mus., i. 117; Ib., v. 19; Ralph, ii. 80; Deveze, p. 63; Ffirth, p. 36; Harrison, ii. 139; Kelly, xiv. 382; Rochoux, p. 531; Dubreuil, viii. 329; Fever of Cayenne (1850), p. 163; J. Davy, Edinb. Journ., lxxii. 280; Blair, p. 93.

⁵ Physick, v. 132; Blair, p. 92; Lawrence, x. 257; Nott, ix. 279; Louis, p. 97; Kelly, xiv. 382.

⁶ Louis, p. 97; Rush, pp. 90, 92; Deveze, p. 63; Kelly, xiv. 382; Rochoux, pp. 354, 531–32; Hayne, vi. 344; Ib., vii. 13; Nott, Am. Journ., ix. 279; Imray, liii. 90; Caldwell (1805), 99; Maher, p. 884; Evans, p. 316; Fev. of Cayenne (1850), p. 163; Chambole, xiii. 201; R. Arnold, pp. 317, 18; Blair, p. 93.

⁷ S. Jackson, p. 21; Rush, iv. 44; Cartwright, ix. 41; Townsend, p. 190; Ticknor, N. A. Journ., iii. 231; Imray, liii. 90; Kelly, xiv. 382; Louis, p. 97; Maher, p. 884; Bally, pp. 352, 366; Evans, p. 316; Rochoux, pp. 354, 531; Hayne, vii. 9; Pariset, p. 352; Copland, iii. 143; Fev. of Cayenne (1850), p. 163; Hackett, xvi. 290; Pinkard, ii. 226; Blair, p. 92; Lawrence, ix. p. 7; Ib., x. pp. 5, 7, 9, 10, 257; Hayne, vi. 344; Ib., vii. 13.

⁸ Kelly, xiv. 382; Maher, p. 884; R. Arnold, pp. 317, 18; Louis, p. 97; Arejula, pp. 426, 7, 8; Rochoux, p. 532; Audouard, p. 155; J. Davy, Ed. Journ., lxxii. 280.

⁹ Med. Mus., v. 20, 21; Townsend, p. 192; Barton, p. 40; Deveze, p. 67; Kelly, xiv. 282; Pugnet, p. 363; Riseuno, in Burnett, pp. 242, 3; Bally, p. 293; Maher, p. 845; Palloni, p. 11; Pariset, p. 352; Rochoux, p. 531.

nant or congestive character, an effusion is discovered under the tissue in question.

These changes are not all found in the same cases; nor are they present at whatever period of the disease the patient may have succumbed; or whatever may have been the character and duration of the disease. In congestive or malignant cases, we find more or less injection of the capillaries—the redness being generally of a dark hue. There are usually ecchymosis and petechiæ; but no thickening, softening, or similar changes. These, together with capillary injection, are, as a general rule, met with after attacks of a different character. In these, if death takes place on the second, third, or fourth day, the increased vascularity is noticed in bright red or dark, dusky patches, more generally confined to the vicinity of the orifices, but sometimes extending to the greater part of the membrane. If the case has been protracted to a later period—to the sixth, seventh, eighth, or ninth day—a larger portion of the surface is found involved, and we may expect to find it of a leaden, livid, greenish, or mottled appearance, and presenting the marks of disorganization already noticed.

In instances unattended with these changes, in which there is mere redness, with ecchymoid and petechial spots, these are probably not the effect of cadaveric changes, for they are found immediately after death, and too soon to be attributed to such a cause. They must rather be referred to simple congestion. But whenever this redness is attended with thickening, or softening, or the mammillated appearance of the membrane, we cannot but join in opinion with Louis in attributing these changes to an inflammatory condition of the parts.

It may be proper to remark that these changes are not always found to prevail in the same proportions; for, while Louis found the mammillated appearance in two-thirds of the cases, thickening in one-half, and softening in many, the latter were not seen by Ruz (p. 17) or Harrison (p. 139); and Kelly says they seldom were found at Mobile (p. 382).

Œsophagus.—The appearances found in the œsophagus are not very unlike those noticed in the stomach. The mucous membrane is often, though not necessarily, altered in colour. When this is the case, it is blackish, deep brown, more or less red, or of the colour of onion parings. In most cases, it is of natural thickness and consistence; but in others it presents unequivocal signs of inflammation and congestion, and not unfrequently it is abraded—the epithelium having, to a greater or less extent, disappeared. This is more common when vomiting has been very severe.¹ This diseased condition often extends to the pharynx.

Intestines.—The changes discovered in the small intestines do not differ materially from those exhibited in the stomach, except that they are generally less marked; they are, perhaps, more frequently absent in the former than in the latter. Externally, the intestines are often of a yellow colour (*Rochoux*, p. 358; *Pugnet*, p. 363), with patches of red, rose, brown, or slate hue, or

¹ Louis, p. 78; Blair, pp. 93–101; Gillkrest, ii. 276; Waring, p. 53; Kelly, xiv. 382; Bache, xxvii. 122; Evans, p. 220; Lawrence, x. 6–8, 253–256; McArthur, p. 349; Dubreuil, viii. 329; Rochoux, xxiii. 253; Belcher, xxiii. 253.

even of black. But, though these appearances proceed often or generally from diseased changes in the peritoneal covering of the parts, they are not unfrequently due to the morbid condition of the mucous or lining membrane of the tube.¹ This morbid condition of the membrane in question is attributable to the same diseased process that gives rise to kindred changes in the mucous lining of the stomach—simple congestion of the capillary vessels, or, as sometimes occurs, and is generally believed, inflammation. The discoloration is sometimes extensively diffused, or, as perhaps is more generally the case, it exists in patches of greater or less size. It occupies, on the whole, between one-tenth and one-sixth of the entire surface of the membrane, and assumes the character of the rouge *pointillé* or *picté* noticed in the stomach; while, sometimes, the membrane is thickened, or softened, or ulcerated, or denuded and even gangrened.

As regards their contents, these vary in character and appearance. When the case has been rapid, they are often found bilious, yellow, and almost always entirely excrementitious.² Under other circumstances, they are brown, black, thick or jelly-like, often of a tar-like appearance. At other times, they are fluid, of a reddish or soot colour, or even consist of blood, more or less pure. Sometimes they are whitish, and often they present the coffee-ground characteristics of the black vomit. On the latter subject, enough has been said in a former chapter.

Of the small intestines, those most generally affected are the duodenum and the upper portions of the jejunum; though, in other cases, the lower portions of the ileum are more implicated than the latter.³

In not a few instances, the intestines are found contracted, to a greater or less extent, and with more or less force; and present, more frequently during some epidemics than others, extensive intussusceptions or invaginations. In one of four bodies examined by the late Dr. Stuart, in 1805, he found six, and in another, three of these intussusceptions, without any signs of inflammation.⁴ They were frequently found, during the same season, by Dr. Caldwell (p. 100) and Dr. Parrish.⁵ In Mobile and New Orleans, they are commonly noticed, according to Dr. Kelly and Dr. Harrison; the latter of whom informs us that in 1839 they were uncommonly so—the quantity invaginated exceed-

¹ Chisholm, i. 183; S. Jackson, p. 79; Ffirth, p. 36; Med. Mus., i. 117; Thomas, p. 132; Rush, iii. 92, 93; Barton, p. 40; Deveze, p. 63; Lawrence, x. 4, 5, 256; Bancroft, p. 39; Hunter, p. 161; Pugnet, p. 363; Gillespie, p. 70; R. Jackson, Tr., p. 265; Waring, p. 53; McArthur, p. 349; Ralph, ii. 80; Desportes, i. 202; J. Davy, Edinb. Journ., lxxii. 280; Caillot, p. 166; Rufz, p. 17; Nott, ix. 280; Louis, pp. 100, 106, 107; Palloni, p. 11; Catel, p. 12; Maher, p. 885; Harrison, ii. 139; Belcher, xxiii. 252; Pariset, pp. 353, 366; Jackson, Sketch, 62, 63, 94; Frost, xiii. 253; Savarésy, p. 461; Dubreuil, viii. 329; Arejula, p. 426; Caldwell (1805), p. 99; Kelly, xiv. 383; Vatable, p. 348; Cayenne (1850), p. 164; Copland, iii. 144; Chambolle, xiii. 201; Ramsay, Charleston Journ., ii. 637; Hayne, ib., vii. 348; Blair, p. 94; Bache, xxvii. 123.

² Rochoux, p. 359.

³ Evans, p. 219; Gillkrest, ii. 276; Harrison, ii. 139; Bache, xxvii. 122; Rochoux, pp. 359, 534; Ffirth, p. 36; Blair, p. 95; Hayne, vi. 484.

⁴ Med. Mus., ii. 305; Rush, iv. 97.

⁵ Med. Mus., iii. 188, 189.

ing sometimes a yard. They were also noticed by Dr. Copland (iii. 143), and have occasionally been seen by the author of this volume.

The glands of the intestines, though less frequently or extensively affected in the yellow than in some other forms of fever, are nevertheless, especially those of Brenner, occasionally found in a diseased or abnormal condition. In some instances, they present a miliary aspect.¹ Those of Peyer have only been discovered altered in cases of a typhoid character (*Kelly*, p. 382; *Harrison*, p. 139), in which, according to Dr. Harrison, there existed before death a low nervous delirium. In some epidemics, as that of Gibraltar in 1828, they were invariably found uninvolved in the disease.² The mesenteric glands are sometimes tumefied. The last mentioned author found them so when death had occurred after the seventh or eighth day, or in cases treated on the mercurial plan. Dr. R. Arnold found them much enlarged, from four inches below the duodenum (p. 318).

In some cases, the mucous membrane of the large intestines is of a bright red, or grayish, or yellowish colour. In the greater number, however, it is pale or white, or nearly so. Its consistence is often diminished. According to Louis, this takes place in two-thirds of the cases. Sometimes, but seldom, it is thickened.³ It has been found decidedly inflamed and ulcerated;⁴ but this change is less frequent in this organ than in the stomach.⁵ The intestine is sometimes contracted, and frequently its internal surface is lined, especially the colon, with a dark or blackish pultaceous matter. The latter is sometimes entirely black; in other instances it is brown, or of a chocolate colour; in others, again, the contents of this intestine consist of a pale red fluid, like blood, or even of pure blood, the surface being smeared over with a substance resembling linseed meal and water.⁶

BILIARY ORGANS. *Gall-Bladder.*—The gall-bladder is sometimes found diminished in size, or even shrunken, and, as it were, withered, and empty.⁷ In other cases, on the contrary, it is distended, and contains the usual quantity of bile of a more or less natural quality.⁸ Again, the fluid is at times in small quantity, viscid, inspissated, or mixed with more mucus than common. It is either dark green, blackish brown, or of an obscure red colour, and of the consistence of tar; and the organ not unfrequently contains a quantity of thick viscous blood, grumous, tar-like, or ink coloured,⁹ or of serum, and more rarely of pus (*Gillkrest*, ii. 274).

¹ Harrison, ii. 139; Rufz, p. 17; Ramsay, *Charleston Journ.*, ii. 637; Blair, p. 94; Copland, iii. 144.

² *Cyclop.*, ii. 276.

³ Louis, p. 111.

⁴ Belcher, xxiii. 252.

⁵ Nott, ix. 280; Rochoux, p. 359.

⁶ Louis, p. 11; Kelly, xiv. 382; Nott, ix. 280; Rufz, p. 17; Gillkrest, ii. 276; Pariset, p. 354.

⁷ Frost, xiii. 253; Pariset, p. 356; Gillkrest, ii. 274; Catel, p. 12; Copland, iii. 143.

⁸ Harrison, ii. 138; Ffirth, pp. 36–7; Ralph, ii. 81.

⁹ S. Jackson, p. 78; Rush, iii. 92; Deveze, 65; Palloni, p. 8; Copland, iii. 144; Harrison, ii. 138; *Fever of Cayenne* (1850), p. 164; Kelly, xiv. 383; Rochoux, pp. 362, 536; R. Arnold, p. 317; Frost, xiii. 253; Blair, p. 97; Dickson, p. 357; Hayue, vii. 16.

The internal membrane is often found spotted or punctated, and sometimes largely injected with blood of a bright or obscure red or brown, or even dark colour;¹ and by several writers on the fever of both tropical and temperate climates, the tissue has been described as presenting more or less frequently traces of unequivocal inflammation²—at other times as healthy. (*Ffirth*, p. 36.)

Liver.—From an early period this organ has been found to present an appearance very different from that of health, or from that it exhibits in other febrile diseases. Unlike the liver of bilious remittent fever, which is usually of a bronze hue, that of the yellow fever is described as being of a light yellow, nankeen, fresh butter, straw, coffee and milk, gum yellow, buff, gamboge, light orange, or pistachio colour. Desportes tells us that the liver is filled with a humour of a whitish or yellowish colour (i. 254).

Moultrie, who came a short time after, informs us that the abdominal viscera, and among them the liver, are of a yellow colour (p. 24). Hume says of that organ that, “instead of being of a dark red colour, as it naturally is, [it] was of a pale yellow, resembling those parts of the stomach and colon in contact with the gall-bladder” (p. 206). Chisholm speaks of the liver being shrunk to less than one-half its natural size—“uncommonly flaccid, and of a colour nearly approaching to buff, or a mixture of yellow and that of ashes” (i. 183–5). Rouppe found it “quite yellow, as if it had been boiled, and almost entirely destitute of blood” (p. 416). “*Hepar bis flavum, sanguine fere in totum vacuum, cocto multum non dissimile, deprehendi*” (p. 310). McColme, the results of whose dissections are recorded in Dr. John Hunter’s work on the diseases of the army in Jamaica, and who served in the West Indies in 1741 and 1742, says: “In all the cases, the liver was changed in part (and sometimes almost the whole), to be more pale and hard than natural; and in such parts there was a less proportion of blood than in those of a more natural colour” (p. 160).

Kindred observations have been made by almost every writer who has paid attention to the anatomical characters of the disease.³

But, though this change in the appearance of the liver was known and had been described over and over again long before the epidemic of Gibraltar in 1828, the attention of the profession has been more particularly drawn to it since the publication of the medical history of that event by Louis, who found

¹ Harrison, p. 138; Kelly, xiv. 383; Vatable, p. 348; Hayne, vii. 16.

² S. Jackson, p. 78; Blair, p. 97; Rochoux, pp. 361, 536; R. Arnold, p. 317; Evans, p. 220; Waring, p. 48; Ruz, p. 28; *Ib.*, Archives, Sept. 1839, p. 73; Barton, xv. 40; Catel, p. 12.

³ Rochoux, pp. 363, 536; J. Davy, *Edinb. J.*, lxxii. 280; Imray, liii. 91; Pascalis, *Repos.*, iii. 348; Lowber, v. 20; Lawrence, x. 4, 8, 11, 254; Parrish, *Mus.*, iii. 188; Ralph, ii. 80; Townsend, p. 190; Gillkrest, ii. 275; Dubreuil, viii. 330; Maher, pp. 852, 885; Catel, p. 12; Pariset, pp. 355, 366; *Ib.*, *Obser.*, p. 18; O’Halloran, p. 186; T. Smith, xxxv. 45; Audouard, p. 73; Blair, p. 97; Barrington, xii. 316, 322; Shattuck, *Transl. of Louis*, xxiii; Costa, p. 84; Barry, *Med.-Ch. Rev.*, xii. 540; *Fev. of Cayenne* (1850), p. 164; Copeland, iii. 144.

it in all the dissections he made on the occasion, and, from the constancy of its appearance, was led to regard it as peculiar to the disease in question (pp. 117 and 162). The more recent dissections made in this city and country, have fully confirmed the statements of Louis and others as to the very frequent, if not constant, occurrence of this discoloration.¹ In some cases, this discoloration occupies the whole surface and pervades the entire parenchyma of the organ; while, in others, it extends only partially over both, giving thereby to the organ a marble appearance—presenting throughout patches or regular striæ, and alternating with others of a dark green colour. It is limited occasionally to a single lobe, usually the left.

Frequently, however, as this peculiar coloration of the liver has been observed, it is far from being universally so; cases occurring in which the organ is found of a different hue—dark yellow, brown, red, purple, bluish, slate, chocolate, or livid.² It has been described as of brick colour, and compared to rhubarb, or to Peruvian bark (*Arejula*).³ In other cases, again, it retains its natural appearance externally and internally, and is otherwise healthy.⁴ The parenchyma, when divided, is often found hard, dry, tough, and sometimes dry and brittle, and more or less devoid of blood;⁵ while, in some cases, the viscus is more or less gorged with blood, and softer in texture than natural.⁶

In some cases, the biliary pores contain bile, but more frequently there is no indication of biliary secretion. Gillkrest, who appears to have examined the subject with attention at Gibraltar in 1828, states that portions of the liver, washed, pressed or bruised in a mortar, did not give out colouring matter, whatever the shade might have been; and portions of the light olive-coloured remained unchanged after long maceration in spirits of wine. No

¹ Bache, p. 121; Hayne, vii. 15; R. Arnold, p. 317; Stewardson, Am. J., N. S., iii. 94; Ash. Smith, Tr. of N. Y. Acad. of Med., i. 65; Kelly, xiv. 383; Nott, ix. 278; Harrison, ii. 138.

² R. Jackson, Tr., p. 264; Lawrence, x. 8; Kelly, xiv. 383; Nott, ix. 278–9; Harrison, ii. 138; Evans, p. 220; Gillespie, p. 70; Frost, xiii. 253; Savarésy, p. 460; Gillkrest, ii. 275; Palloni, p. 10; Hackett, xvi. 290.

³ Pariset, p. 355; Ralph, ii. 81; Costa, p. 84; Rochoux, p. 535; Blair, p. 97; Chambolle, xiii. 201.

⁴ Rush, iii. 93; Deveze, p. 65; Ffirth, p. 36; Parrish, iii. 188; Lawrence, x. 256; Nott, ix. 279; Rochoux, p. 363; Ramsay, Charleston J., ii. 637; Maher, pp. 885.

⁵ Lawrence, x. 6, 8, &c.; Waring, p. 48; Gillkrest, ii. 275; Pugnet, p. 363; Desportes, i. 204; Frost, xiii. 253; Nott, ix. 278; Kelly, xiv. 383; Harrison, ii. 138; Blair, p. 96; Arcjula, p. 426–8; Maher, p. 885; Dubreuil, viii. 330; J. Davy, Ed. J., lxxii. 280; R. Arnold, p. 317; A. Smith, Tr. of N. Y. Acad. of Med., i. 65.

⁶ Lawrence, x. 13; Kelly, xiv. 383; Vatable, p. 348; Jackson, Tr., p. 264; Pugnet, p. 363; Evans, p. 22; Gillespie, p. 70; Jackson, Sketch, i. 64, 72, 77; Ralph, ii. 81; Savarésy, p. 460; Nott, ix. 278; Gillkrest, ii. 274; Harrison, ii. 138; Rufz, p. 18; Waring, p. 48; S. Jackson, p. 79; Merrill, ix. 246; Cartwright, ix. 40; Ffirth, p. 36, and Mus., i. 117; Lowber, Mus., v. 19, &c.; Mitchell, Mus., i. 3; Barrington, xii. 314; Desportes, i. 202; Pariset, pp. 48, 51; Frost, xiii. 253; Barton, xv. 40; Ticknor, iii. 29, 31; Smith, Am. J., xxv. 506; Hulse, of Pensacola, quoted by Nott; Deveze, p. 65; Fever of Cayenne (1850), p. 164; Doughty, pp. 106–7; Copland, iii. 144; Chambolle, xiii. 201; Dickson, p. 357.

trace of bile was observed in the pores on the occasions just referred to, nor was the hepatic or common duct found obstructed, like the cystic (*Gillkrest*, ii. 275). The same results have been obtained in this city.

Though often more or less enlarged,¹ and at other times shrunk,² the liver seldom exhibits traces of inflammation, and, if these are discovered, they must be viewed simply as the effect of complications; while the alteration in, or suppression of, the secretory function of that organ, may justly be referred to some cause different from that morbid state.

Dr. Harrison remarks of the appearances which the liver exhibits, that the latter is subject to them in common with all the organs; that "the existence of one or the other appears to depend much upon the condition of the patient at the time of the attack and the treatment he has undergone;" and that, "in cases in which the lancet has been used freely, we shall generally find a pale yellow liver" (p. 138).

The pale yellow liver is more common and marked in women and children; in the latter of whom it assumes, after a short maceration, the paleness of boxwood (*Cycl.*, ii. 275). It differs, as regards frequency, in different seasons, and even at different periods of the same season. In Gibraltar, during the epidemic of 1828, it was very common; so much so, indeed, as to be regarded by Louis as universal. Pascalis speaks of it as general in New York in 1822; Catel found it constantly in the one hundred and fifty dissections he made in the Military Hospital of Martinique in 1838-9. It was found in this city in almost every case examined in 1853 and 1854. On the other hand, Dr. Nott perceived it, at Mobile, in only two cases out of eight in 1843, and in four out of the same number in 1844 (p. 278). Maher speaks of it as occurring in only one-half of the cases dissected by him at the Havana (p. 885). The organ in the others was healthy. During the latter part of the epidemic season of 1828 at Gibraltar, the pale yellow colour gave place to a reddish-brown, compared by some to that of Peruvian bark, and by others to the leaves of an autumnal season.³

In the second edition of his important work on the liver, Dr. Budd, after remarking that the entire liver may be damaged by some acute disease, or in other ways, and may become fatty in consequence, threw out the suggestion that this may happen in yellow fever and in the severe bilious remittents of tropical climates. "These fevers," he said, "without leaving any permanent marks of inflammation, and apparently without exciting inflammation at all, may permanently alter the condition of the liver. It often happens that the office of the liver is not adequately performed for the future, and that years after, when the person dies, perhaps from some disease quite independent of this, the liver is found unusually pale. The pale colour of the liver depends, I imagine, on fat, which is not present, however, in such quantity as to increase the size of the liver, and to cause the striking appearances of the extreme fatty liver of phthisis" (p. 300).

¹ Louis, pp. 117, 162; Evans, p. 220; Maher, p. 885; Frost, xiii. 253.

² Chisholm, i. 183.

³ Gillkrest, *Cycl.*, ii. 275.

A few facts observed antecedently to the publication of Dr. Budd's volume, but evidently unknown to him, and many collected subsequently in this country by pathological inquirers, who at the time had not seen that volume, go far to confirm the views of its author, so far at least as they relate to the yellow fever. In one of the cases mentioned by Sigaud, as having occurred sporadically in Rio Janeiro during his residence in that city, the liver was found hypertrophied; and all that part of its superior lobe which corresponds to the diaphragm was found to present "the first degree of fatty degeneration."¹ More recently, Professor Alonzo Clark, of the College of Physicians and Surgeons of New York, called attention to the microscopical examination of the liver of a patient who had died of yellow fever in the New York Hospital in April, 1853. The individual had arrived from some place on the Gulf of Mexico, where the fever then prevailed. The symptoms were such as to leave no doubt as to the nature of the disease, and the diagnosis was confirmed by the *post-mortem* appearances. The microscopical examination of the liver, which the author of these lines had the opportunity of witnessing, demonstrated "a fatty state of all the secretory epithelial cells, and an abundance of free fat globules."² Dr. Clark would not, from this single observation, conclude that the lesion thus discovered was peculiar to the yellow fever, for he felt that, in the case which presented it, the liver might possibly have been fatty before the attack of the disease which carried off the individual. He therefore contented himself with putting the question to future inquirers: "Is not the change so constantly observed in the livers of those dying of yellow fever an acute fatty degeneration?" Scarcely a few months had elapsed when we had in this city the fullest opportunity of confirming the discovery of our very distinguished and accomplished countryman; for, in all the examinations made during the epidemic of 1853, the peculiar change in the liver to which he called attention was discovered. These examinations amounted to fourteen at the Pennsylvania Hospital. Several more were made at the St. Joseph and other hospitals. In the summer of the next year (1854), the livers of those who died with the disease at the first of these establishments—six in number—were in like manner examined under the microscope; and *in every instance, without a single exception*, in both seasons, the results were the same. The number of livers examined was too large; the results obtained were too constantly the same to allow these to be ascribed to mere coincidence. The changes observed must appertain to the disease, and, as they are not noticed in the livers of individuals who have died of other fevers, they must be placed among the anatomical characters of the yellow fever. The examinations at the Pennsylvania Hospital were made by the resident physicians of that institution, one of whom, Dr. T. H. Bache, has published a record of them. Others were made, at my request, by Professor Leidy, of the University, whose authority in matters of this sort is among the highest in the land. The following letter to me, from that gentleman, contains the results of his observations:—

¹ Climat et Maladies du Brésil, p. 260.

² New York Medical Times for May, 1853, p. 238.

“The portions of livers which you submitted to me for examination, from nine different persons who had died of yellow fever, presented a uniform pathological condition.

“The colour of the pieces varied from a yellow clay-like hue to a brownish orange, becoming darker on exposure to the air from drying, and thus rendering the blood more visible. The pieces, also, were a little less firm than usual; and, when small detached fragments were dried on paper in the sun, widely-extended grease spots resulted. Beneath the microscope, the structural elements of the liver presented a similar appearance as in fatty degeneration of the organ in drunkards, and some cases of pulmonary disease. The hepatic secreting cells were unaltered in form and structure from the normal condition; but they differed in having desposited in their interior a variable amount of oil globules, frequently entirely obscuring the nucleus, which, however, was rendered readily visible on the application of acetic acid. The oil globules varied in size from a small granule to the one-half the diameter of the containing cells, and many of those observed loose in the field of the microscope, which had escaped from lacerated cells, ran together and formed drops larger than the cells. I think the uniformity with respect to the presence of so much fat in the liver of these yellow fever cases, clearly indicates it as one of the pathological phenomena of the disease, and, although it may throw no light on the character of the affection, yet I deem it an important fact, to be added to that accumulative mass of evidence by which medical science gradually, but surely, moves onward.”

The following are the appearances found in the hospital cases: “The secreting cells were pale, ill-defined, and less granular than when in the normal state. In the cells, with few exceptions, no nucleus could be detected, but its place was supplied by a single oil globule. This was observed even in those cases in which the granular part of the cells was not so full of oil as in some others. Generally, the cells were so studded with oil globules as to give one the idea of looking at a number of the latter, which had by chance become agglomerated, or entangled by granular matter, leading to the conclusion that the cells were broken down. I am very sure we at first mistook some diseased cells for such oil globules. Nor did the oil globules confine themselves merely to the granular part of the cells and their nuclei, but they were found floating freely, of various sizes, all over the field of the microscope.”¹ In none of the examinations made at the hospital, or by Dr. Leidy, could any difference be detected between the liver of yellow fever and that of drunkards and of consumptives, while the peculiar colour of the organ noticed in the former disease, was proved to depend on the presence of the oil; inasmuch as, in every portion of the substance in which the alteration of colour had taken place, oil-globules were found, while none were discovered in places where no alteration was noticed.

Prof. Riddell, of New Orleans, has been less successful in his researches in this matter. In a recent pamphlet, *Microscopical Observations pertain-*

¹ Bache, Am. Med. J., July, 1854, xxvii. 124-5.

ing to *Yellow Fever*, he informs us that, by means of the *post-mortem* researches of Drs. Macgibbons, T. Hunt, and Fenner, he has had "opportunities of examining about fifty human yellow fever livers, and that fatty degeneration, well-marked, occasionally appears, but not constantly." Dr. Riddell has seen it in chronic dysentery and other diseases, and thinks that it is a far less constant lesion in fatal cases of yellow fever than the molecular change he discovered in the heart. Further observation, by competent hands, must decide the matter. In the meanwhile, we must regard the constant occurrence of the pathological condition in question, in the cases examined under my eyes by such experienced observers as Drs. Clark and Leidy, and the resident physicians of the Pennsylvania Hospital, as justifying the conclusion drawn therefrom.

Kidneys.—These organs are not unfrequently found in a normal state, or with only trifling marks of having partaken of the disease. Such was found to be the case by Dr. Deveze during the epidemic of this city in 1793, and Ffirth (p. 36) in 1802, and judging from the silence of Dr. Jackson, in his account of the fever of 1820, as to the condition of these organs, we may presume that the result was the same that season. The examinations made during the epidemic of 1853, and the less severe visitation of 1854, led to nothing of importance in respect to those organs. Dr. Ralph says, in reference to the fever he observed at Barbadoes, "no unnatural appearance was discovered in the kidneys, although they were always minutely examined" (p. 81); and other testimony as regards the normal state of these organs in the fever of tropical and temperate climates, may easily be found.¹

In ninety-seven dissections made by Dr. Blair at Georgetown, Demerara, in 1841, '42, and '43, he found the kidneys diseased in forty-four cases, or one-half (p. 98).

In other cases they are congested—filled, more or less, like other organs with dark fluid blood, the mucous membrane of the pelvis and infundibulum being sometimes minutely spotted with blood, or ecchymosed; or they exhibit a morbid state, similar to that observed in Bright's disease.²

According to Pennell, not only are the organs congested, but the papillæ yield upon pressure a glutinous tenacious exudation, sufficient to interfere mechanically with the escape of urine (*op. cit.*, p. 252). At Cayenne, in 1850, the kidneys were found to have lost a portion of their consistence. The cortical substance was softer than natural, or engorged with diffuent blood, and contained a lactescent fluid, and, in many cases, pus. The serous membrane and fat were yellow. The ureters and pelvis sometimes had pus (p. 164).

In others, again, they are found to bear the marks of acute inflammation.

¹ Rochoux, p. 537; Dubreuil, viii. 321; Louis, p. 142; Arnold, p. 25; Bally, p. 244; Harrison, p. 138; Pariset, pp. 356, 7; Waring, p. 48; Frost, xiii. 253; Chambolle, xiii. 201, 2; R. Arnold, p. 318.

² Rufz, p. 19; Harrison, ii. 138; Vatables, p. 348; Chambolle, xiii. 201, 2; Pennell, *Med.-Ch. Tr.*, xxxvi. 252; Davy, notes to Blair, p. 98; *Ib.*, *Edin. Journ.*, lxxii. 281; Blair, p. 98; *Fever of Cayenne* (1850), p. 164.

Savarésy (p. 461) describes them as being always inflamed and engorged, sometimes sphacelated. Catel found, in some instances, the tubular substance in a state of inflammation (p. 12). Kelly says, that the substance of the organ is occasionally congested with minute abscesses in the papillæ and pus in the ureters (p. 383). Pathologists in the West Indies,¹ some in this country,² and others in Europe,³ found those organs inflamed. "It is the general opinion," says Dr. O'Halloran, "that this viscus is not liable to suffer from inflammation and its consequences in the yellow fever even when suppression of urine is a fatal and prominent symptom for days. I have seen more than 200 dissections, but I do not recollect having seen the kidneys examined minutely; a single incision through the pelvis satisfied the examiner, who pronounced it healthy." At first, Dr. O'H. was equally careless, and the results were the same. At last, in a case which he dissected, he "accidentally discovered abscesses," and this led to the examination of the ureters, which also contained pus. "I cannot say," he adds, "that abscesses of the kidneys are common in the yellow fever of Andalusia, having only performed a few dissections there; but as the symptoms of the disease of Barcelona (where I am disposed to think, from what I have witnessed, that abscesses of the kidneys were common) bore a considerable analogy to the inflammatory form of the Andalusian fever, I am inclined to believe that the same appearances exist, and will be found on dissection" (pp. 200, 201).

Bladder.—This organ is often found contracted.⁴ In some cases, its coats are thickened.⁵ Though mentioned as sometimes inflamed,⁶ the mucous membrane is more generally healthy, or only injected and dotted with small points of blood, or ecchymosed; while, in some cases, it is lined with yellow mucus. It is often nearly, if not quite empty;⁷ in some instances, on the contrary, it contains a variable amount of urine, which sometimes is natural in appearance and quality, and at others, more or less bloody. In other cases, again, the organ contains black matter resembling that ejected from the stomach, or pure blood.⁸

Spleen.—This organ is generally found of a darker colour than natural—sometimes somewhat enlarged and friable. It is often moderately softened, and generally engorged with dark currant jelly-like blood.⁹ In some cases it is found unchanged.¹⁰

¹ Rochoux, p. 364; Pugnet, p. 363; Blair, p. 98.

² Gros, pp. 19, 20; Merrill, ix. 246; Waring, p. 48; Cartwright, ix. 42; Lawrence, x. 256.

³ Lasso, Caisergues, pp. 175, 183; Palloni, p. 9.

⁴ Deveze, p. 66; Dickson, p. 357; Blair, p. 98; Copland, iii. 145; Kelly, p. 383; Ffirth, p. 56; Rochoux, p. 364; Ralph, p. 81.

⁵ Kelly, xiv. 383; Blair, p. 98.

⁶ Ffirth, p. 36; Catel, p. 12; Dickson, pp. 357, 365; Rochoux, p. 537.

⁷ Copland, iii. 145; Fever of Cayenne (1850), p. 165.

⁸ Kelly, loc. cit.; Harrison, ii. 138; Nott, ix. 280; Rufz, p. 19; Deveze, p. 66; Dickson, p. 357; Fever of Cayenne (1850), p. 165.

⁹ Louis, pp. 141, 162; Rochoux, pp. 365, 538; Ralph, p. 86; Copland, iii. 143; Hayne, vii. 17; Fever of Cayenne, p. 164.

¹⁰ Ffirth, p. 36; R. Arnold, p. 317.

No change, some degree of softness, perhaps, excepted, has been detected in the pancreas.¹

In some cases, the penis is found covered with eschars, and the scrotum swollen and thickened—brown or black, as in senile gangrene—sometimes with excoriations.²

Dr. Hayne states that in the uterus of every female examined by him in 1849, at Charleston, he found blood; and, in the ovaries of each, either a coagulum or positive traces of hemorrhage. He was satisfied, by careful observation, that none of those females were menstruating at the time of the attack, or during their sickness, as a proof of which it is stated that no traces of blood were found in the vagina, or at the os tincæ after death.³

CHAPTER XVIII.

ANATOMICAL CHARACTERS OF YELLOW FEVER—CONNECTION OF CHANGES FOUND ON DISSECTION WITH SYMPTOMS.

HAVING now passed in review the morbid changes discovered on dissection in the different organs and tissues, it becomes necessary to examine briefly whether it is possible to discover any that appertain, in a special manner, to the yellow fever, and serve to distinguish it from other febrile diseases; in other words, whether dissection reveals any anatomical characters specially belonging to the former, and imparting to it a distinct nosological position. To a certain extent, the discoloration of the skin, usually discovered after death, might be regarded in the light of a distinct anatomical character, as it shows itself in some of its various shades much more frequently in this than in other fevers. But, on the other hand, it loses its claims to be so considered, from the fact that it does not exclusively appertain to the disease in question, and is, besides, absent in some cases, the yellow fever character of which cannot be doubted. Neither can we discover in the brain, or its membranes and dependencies, anything characteristic of the disease. Inflammation or congestion of these parts, especially of the first, are far from being always present; and if they were, would not be entitled to be held in the light in question, inasmuch as the same morbid changes are common to many other and very dissimilar complaints. Of the morbid condition of the ganglionic centres, on which much has been said by Dr. Cartwright, we know too little to justify our raising it to the rank of an ana-

¹ Ffirth, p. 36; Copland, iii. 144.

² Pariset, p. 358; Arcjula, p. 160.

³ Charleston Med. Journ., vii. 17. See Michel, same work, v. 748, 9.

tomical character, particularly as changes, similar to those he describes, are occasionally found on the dissection of individuals who have died of other diseases, and as, on the other hand, in yellow fever itself, they often fail to present themselves. The lungs, heart, œsophagus, gall-bladder, kidneys, and bladder exhibit nothing peculiar, or that is unobserved in other complaints. The petechial-looking spots discovered on the surface of the first of these organs, and the fibrinous coagula found in the heart, may perhaps form exceptions to this statement, as they are found oftener in yellow fever than in other kindred or dissimilar complaints. Nevertheless, they are not always found in the former, and are sometimes, if not often, seen in the others; and it would be hazardous, therefore, to pronounce an individual to have died of yellow fever from the mere discovery of those changes. The stomach itself, though considered from the days of Warren as the throne and seat of the disease, presents nothing which can be regarded so strictly peculiar as to enable us to identify the disease. Inflammation of its lining membrane, and the injection from simple congestion of its vessels—whatever shape the latter may assume—though generally found, are not unfrequently wanting, the former more especially; and we all know that these conditions of the same parts are discovered just as often in other cases which can in no way be confounded with the yellow fever. Nor can we even regard the red or black fluid matter discovered in the stomach, common as it may be, as an unerring anatomical character of the disease; since it is not found in all cases, and, on the other hand, shows itself in other affections in no way allied to the yellow fever. It is, therefore, not peculiar to the latter.

Relying on the results of his and other observations at Gibraltar, in 1828, where, as we have seen, the liver presented a light yellow colour, both externally and internally, and was, besides, dry and tough, Louis concluded that the above-mentioned alteration of that organ should be regarded as the anatomical character, *par excellence*, of the disease (p. 163). Elated, apparently, with the idea of having made, as he thought, a discovery in relation to the peculiar appearances and morbid condition of that organ—a pretension he would not have claimed had he not in this, as in other matters, neglected to extend his reading beyond his own writings and those of his pupils—the French morbid anatomist regarded the fact of the particular discoloration of the liver, as of the highest importance in a diagnostical point of view; affording, as it must, according to him, the surest means of ascertaining the real nature of the disease, distinguishing it from others, more or less allied to it, and enabling us, in the absence of other proofs, to detect without risk of failure, the epidemic existence of the fever. There is little doubt, that if the condition of the organ were always such as is here described, and if it were truly an anatomical character of the yellow fever, it would be entitled to particular consideration, establishing, as it would, a line of demarcation between the fever in question and all others—even the one with which it is most frequently confounded, the bilious remittent, in which the same organ has been found to present a bronze tint, sometimes superadded to a slaty colour, and

other peculiarities not found in the disease under examination, and to which attention will be particularly called in a future chapter.

Admitting that such is the invariable appearance of the liver in bilious remittent fever—a fact which, as some think, requires confirmation—the claims of the yellow coloration and dry condition of that organ to being regarded as true anatomical characters of the yellow fever, and to their being sure instruments of diagnosis, fade away somewhat when we find, what has been shown in the preceding pages, that, whatever may be the result in most instances, and especially during some epidemics, the liver not unfrequently presents a very different appearance; while, on the other hand, the yellow liver has, as we learn from Dr. Chervin, Dr. Nott, and other high authorities, been seen in the intermittents of hot climates, and in the bilious fevers of our southern States. In the present state of our knowledge, all we can say on the subject is, that the peculiar discoloration of the liver found in yellow fever, and the fatty condition of the organ to which that discoloration is due, are more frequently noticed in that disease than in others, and approach nearer to what may be regarded as true anatomical characters of the former than any morbid change we have noticed.

Reluctant, however, as we may be to regard any of these changes, when considered singly, as characterizing the yellow fever, we can have no hesitation in admitting that when several of them—a dark mahogany colour of the skin; petechial condition of the gastric mucous membrane; black matter in the cavity of the stomach and bowels; a yellow discoloration and fatty degeneration of the liver; an albuminous state of the urine, &c.—are found associated together in the same case, there will be no difficulty in forming an opinion as to the nature of the disease.

In the yellow fever, more perhaps than in any other disease, it is difficult, if not impossible, to connect as cause and effect the symptoms observed during life with evidences of textural changes discovered after death. That the morbid conditions of the parts, giving rise to the changes in question, must and do manifest themselves during the progress of the disease, by external and recognizable signs, is not to be denied; but it is not the less true that the main and characteristic symptoms cannot always be attributed to any particular discoverable pathological changes in the organs in which they are seated; that they may exist, whether or not those changes are discovered after death, while it may even happen that these are found when the symptoms thought to be due to them had been either absent or of trifling import. On this subject, however, there has existed, and continues to exist, a difference of opinion; and, as it is one of importance, in a pathological and therapeutical point of view, I shall make no apology for offering some facts in support of the position here assumed.

A. *Cerebral Organs*.—It has been remarked, in relation to the yellow fever of tropical climates, that, whenever the symptoms of cerebral affection have existed in a marked degree, the cause may always be detected in the lesions of the brain and its membranes; that the several phenomena which are generally denominated ataxic, are almost always due to inflammation, more or less

intense, of the arachnoid membrane, combined generally with sanguine engorgement of the plexus choroides, the tela choroides, and the lateral ventricles, as well as with more or less serous effusion in the latter. According to those by whom the opinion is entertained, delirium always exists when this effusion takes place.¹ Dr. Rush, many years ago, referred delirium, as well as suffusion of blood in the face, redness of the eyes, dilatation of the pupils, pain in the head, hemorrhages from the nose and ears, sickness and vomiting, and costiveness, to inflammation and morbid congestion of the brain (iii. 58); and many others have entertained similar opinions, in reference particularly to mental derangements. But, granting that such a condition of the brain and its membranes produces the effects mentioned, when it exists, and that, whenever meningitis is present, delirium will be the consequence, it is a fact, placed beyond doubt, that as frequently, if not more so, delirium and the whole list of ataxic symptoms occur without the possibility of attributing them to any such cause. Dr. Rochoux himself allows that in the West Indies, where, as we have seen, he thinks delirium is due to meningitis, it may, when occurring on the third or fourth day, be symptomatic, and unconnected with inflammation of the membranes (pp. 313, 314); and, in reference to the yellow fever of Barcelona, he says that, notwithstanding the frequency of delirium, no appreciable sign of inflammation was found in the brain, or cerebellum, or pia mater, or arachnoid (pp. 525, 540, 563). To the same effect we may appeal to the testimony of other writers, in both tropical and temperate climates. Dr. McArthur states that, in five cases out of ten, in which much delirium had existed, the brain did not exhibit any marked appearance of disease (p. 350). Even at the very moment of writing these lines, I had an evident proof of the want of connection between delirium and cerebral or meningeal inflammation, for, at the *post-mortem* examination of an individual whose mind was intensely deranged, no change of any kind was discovered in the brain or membranes. The man was highly jaundiced, and the serum in that organ contained a large quantity of the colouring matter of the bile. The delirium may have been due to this.

For like reasons, I am disposed to doubt the propriety of referring, as is done by some pathologists, the acute pain of the head and orbits to inflammation of the arachnoid.² This pain, as seen, is one of the most ordinary symptoms of the disease, appearing in almost every case; whilst, on the contrary, arachnoiditis is of comparatively rare occurrence. The pain, indeed, appears unconnected with the disordered action of the cerebral organ which occasions delirium, and which is, in very many instances, independent of inflammation; for, though delirium occasionally occurs, it does so much less frequently than the pain in question; whereas, if it were the result of cerebral disorder, whether inflammatory or otherwise, we should find it only in cases in which the latter disorder could be detected, either on dissection, or inferred by the occurrence of delirium. It would follow, also, that, every time we have pain, &c., we would find inflammation after death, and, as a consequence,

¹ Rochoux, pp. 369-373, 563.

² Ibid., p. 271.

delirium during life; for though, as stated, delirium may exist without inflammation, the reverse is not true, meningitis entailing delirium or some other cerebral affection. The same remarks are applicable to redness of the face or eyes, which have been supposed to indicate inflammation of the brain or membranes.¹ In many cases, in which these symptoms have existed in a prominent degree, nothing of importance has been detected in those parts. We cannot, therefore, attribute them to cerebral inflammation, when marks of these happen to be detected on dissection.

B. *Spinal Marrow*.—The pain in the loins, and other symptoms, have been referred to the congestion or inflammation of the cord and its membranes, marks of which are sometimes revealed after death, and to the serous effusion often discovered in the canal. On this subject much stress was laid by the French commissioners sent to Barcelona in 1821, who, in a memoir read before the Royal Academy of Medicine in 1823, held up these morbid phenomena, and particularly the serous effusion in question, as the anatomical character, *par excellence*, of yellow fever. It would, perhaps, be improper to deny that, under some circumstances, these morbid conditions may produce some of the effects mentioned; nevertheless, as these symptoms exist, to a considerable extent, in most cases of yellow fever, whilst the cord and its membranes are found diseased in a few instances only; as, on the other hand, the morbid changes exist to as great an extent in other diseases, in which the symptoms in question do not present themselves;² and as the serous effusion is found in combination with a healthy state of the spinal arachnoid,³ and in a variety of diseases, having nothing in common with yellow fever, it is evident we must look to some other cause to account for the phenomena here referred to.

c. *Ganglionic System*.—Much the same objection may be made to the opinion of those who would connect the foregoing and other symptoms with the inflammatory affection of the ganglionic centres and nerves, marks of which they affirm to have detected after death; for these marks are not always found, while the symptoms which the inflammation of these nervous organs are supposed to produce invariably present themselves; and, though the absence of the former cannot invariably be viewed as a certain proof of the non-existence of the inflammation during life, the supposition of the very frequent disappearance of the usual signs of inflammatory changes in the nervous tissues can scarcely be entertained; while, as we have seen, the changes in question are found in diseases characterized by different symptoms.

These remarks have reference to pathological conditions in the ganglionic centres and nerves, capable of leaving such traces behind after death, as will be revealed on dissection. With changes of a vital or functional kind, to which Maher (p. 869), Gillkrest (*Cyclopaedia*, ii. 278), Wragg (x. 77), and others have referred—the pulsation of the cœliac artery; the lumbar pain; the affection of the stomach, bowels, and liver; the suppression or entire

¹ Rochoux, pp. 293-296.

² Pariset, &c., p. 346; Audouard, p. 196; Rochoux, p. 527.

³ Magendie, *Journal de Physiologie*, v. 27, vii. 1, &c.

alteration of the secretions; the arrest of the functions of digestion and assimilation, the tendency to early solution, and even dissolution of the blood; and the heavy, dull, and painful oppression in the whole abdominal region—we have nothing to do at present.

D. *Stomach*.—Influenced by the sufferings of the stomach from the onset of the attack, and the frequent discovery, after death, of marks of inflammation in its lining membrane, many pathologists have regarded the disease in the light of a gastritis—referring all the local sufferings in question to the inflammation of that part, and the other symptoms to secondary affections resulting from the influence of the same. They have thus connected, as effect and cause, the symptoms of the fever with the morbid changes discovered in the stomach. Among those symptoms, epigastric tenderness, nausea and vomiting, and the burning sensation stand prominent. Thus, Dr. Hayne, of Charleston, says that in all the dissections, with scarcely one exception, he found, on *post-mortem* examination, changes in the gastric mucous membrane amply sufficient to explain so well-marked a symptom as the tenderness in question, as also the others mentioned—*i. e.* the softening with inflammation and thickening, and softening without inflammation, either with or without thickening.¹

When, however, we bear in mind that in many cases no traces of inflammation are left after death; that the condition of the parts is generally such as to forbid the idea that inflammation could have existed, and that there is no evidence of its having existed during life and disappeared after death; when we find that in such cases the sufferings of the stomach were much the same as in those in which the appearance of the gastric membrane was different; and when, besides, we recollect that diseases in which the stomach is indubitably inflamed present phenomena very dissimilar to those of yellow fever, the conclusion is natural, that, whatever part the inflammation in question may play in giving rise to morbid phenomena, it is not essential to, or participates but little in, the production of the characteristic symptoms of the disease; and that hence these must be perfectly independent of it, and due to other causes.

The same remarks are, to a certain extent, applicable to the simple congestion of the membrane, though to this morbid state may be referred the exudation which gives rise to the black vomit. Dr. Rochoux has remarked, that the extent of the mucous membrane of the intestinal canal affected in the yellow fever, and the degree of violence of the morbid changes therein produced, are very rarely in relation to, or in harmony with, the intensity of the intestinal pain observed during life (p. 535). The remark is correct; for, in cases characterized by great pain and other intestinal symptoms, no changes, or trifling ones only, are found in the bowels—and *vice versâ*, in instances in which the intestinal sufferings are not considerable, the mucous membrane sometimes bears the marks of having been the seat of considerable inflammation. To the inflamed and denuded condition of the œsophagus, which, as we have seen, is found in some cases, may, it is said, be safely referred the sensation of burning pain and rawness extending all along the canal, and of

¹ Charleston Journ., vii. 9, 10.

which many patients complain bitterly.¹ Here, again, we sometimes find the morbid change disconnected with the symptom attributed to it; for cases, attended with the sensation in question, occur when, after death, the part is not found to present marks of acute inflammation, and no abrasion of the epithelium; and it occurs, also, in cholera, when no such change is found.

E. *Biliary Organs*.—Rochoux attributes the jaundice which occurs in the yellow fever of tropical climates to an inflammation of the gall-bladder and biliary ducts (p. 361), and it is not unlikely that such may be the explanation of some forms of that symptom which, as already stated, is connected with, or arises from, under particular circumstances, some derangement of the biliary function. Nor is there any reason for denying that the morbid changes of the liver described in a preceding chapter, and which usually give rise to jaundice when they occur in other complaints, may and do produce the same effect in the fever before us. In such instances, therefore, there certainly exists a connection as cause and effect between the changes discovered after death in the biliary organs and the jaundiced discoloration of the skin and other parts during life. But the discoloration in question is, as already stated, occasionally of a different nature, and apparently unconnected with biliary obstruction and hepatic derangement; while it occurs in cases where no such obstruction exists, where the gall-bladder and ducts are free from the morbid state mentioned, and where the liver is, so far as can be discovered, either healthy or gorged with blood. Rochoux himself speaks of the vesicle being healthy in most cases observed at Barcelona (p. 536); and we have seen that instances in which the liver presents none of the textural changes described, have, though rarely, been pointed out by reliable authorities. In these, therefore, the connection in question fails, and the jaundice may properly be referred to some functional modification of the liver, the existence of which cannot be discovered on dissection, which exercises its influence in arresting the elimination of the biliary element, whether those textural changes occur or not, and which precede the latter.

F. *Lungs*.—The usual integrity of the lungs—the ordinary absence of inflammation of those organs—leads to the conclusion that the cough which is observed in many cases is merely symptomatic, or the effect of mere nervous irritation; while the oppression, and other phenomena connected with respiratory function, and the indescribable anxiety felt at the præcordia, are due to the same cause, perhaps to the presence of fibrinous clots in the heart; doubtless, in great measure, to the accumulation of blood in the lungs, as well as—perhaps principally—to the unhealthy condition of that fluid.

G. *Heart*.—Dr. Pennell attributes the quick and sudden death that occurs in some cases of the disease before us to the formation of those fibrinous clots either in the heart or large vessels, the presence of which, as we have seen, is often detected on dissection. The idea, he remarks, was suggested to him by his hospital colleague at Rio Janeiro, Dr. Lascaille, who possessed records

¹ Rochoux, pp. 335, 390; Cyclop., ii. 275; Louis, p. 78.

of two hundred *post-mortem* examinations of individuals who had died of yellow fever, and had found the clot in all cases in which the event had occurred suddenly.¹ On this point, I have no experience. To their formation during life he attributes, in some measure, the great distress or fatigue in the breathing, and the indescribable anxiety felt at the præcordia; but principally the physical signs discovered on auscultation, and which have been described in a preceding chapter. Whatever may be the connection between these physical signs and the formation and presence of the clots in question, we cannot justly refer the respiratory distress, and præcordial sufferings, exclusively to this cause; for they exist, sometimes to a very great extent, in almost every case of the disease, whether in those who die or those who recover; whereas, the presence of the clot, as it is supposed, is a sure indication of a fatal issue.

II. *Kidneys*.—By Dr. Waring (p. 48), and Rochoux (p. 299), the pain in the loins is referred to the inflammation of the kidneys, evidences of which are sometimes discovered after death; while, by several writers, the suppression of urine is attributed to the same cause,² or to the existence of abscesses in those organs.³ We have seen, however, that in many cases, both in tropical and temperate climates, the kidneys are found free from inflammation or any other apparent disease; and, as suppression of urine is a symptom of frequent occurrence, we may presume that it often exists without special and appreciable disease of those organs. Indeed, such is mentioned as having been the case by Pariset (p. 356), Rochoux (vii. 554–7), in Europe—by Ralph (ii. 81), Dubreuil (viii. 321), Rufz (p. 19), in the West Indies—some of whom attribute the effect to a mere derangement or cessation of the secretory process, unconnected with appreciable lesion in the organ, the effect of a hitherto unknown pathological condition. This symptom, as well as scantiness and a turbid, serous character of the urine, Dr. Pennell cannot help thinking is due to the partial or entire blocking up by exudation matter of the tubuli uriniferi. “The fluid which escapes evidently contains exudation matter in abundance; and this would account most satisfactorily for the scanty or suppressed secretion.”⁴ On this subject, further and more positive observations are required, and it must be referred to the microscopist.

I. *Blood*.—Are we not justified in explaining, by the changes which occur in the blood of individuals affected with the yellow and other forms of autumnal fevers—changes which are noticed during life and after death—many of the phenomena which present themselves in the course of those diseases?

The following remarks, though having reference to another form of fever, will find an application, as far as they go, to the yellow fever itself:—

“The first action of the poison is apparently upon the functions of the

¹ Med.-Chir. Tr., xxxvi. pp. 246–8.

² Rochoux, p. 364.

³ O'Halloran, pp. 200–1; Daniel, p. 70—to congestion of vessels overpowering the capillaries.

⁴ Med.-Chir. Tr., xxxvi. 252.

blood; those are impaired, or, in peracute cases, cease altogether. The functions of the blood are, first, to maintain the activity of the nervous and muscular systems; and, secondly, to supply the materials for the molecular changes constantly going on in the tissues. It is essential to this being properly performed that the blood-corpuscles be in a fit condition to carry oxygen, and it would appear from the symptoms which mark every stage of fever, that this function of the corpuscles is impaired from the first. This is indicated by the *besoin de respirer* developed in the premonitory stage; the sensation and condition of the respiratory organs are precisely the same as if their capacity had been diminished, and due aeration of the blood thus prevented. The patient takes many forced inspirations, sighs, or gasps, and the breathing is quickened on the least exertion. The hæmato-globuline is changed also, for the skin assumes a peculiarly pale, sallow, and unhealthy look. The function of the nervous system is impaired in consequence of these changes in the blood—namely, the changes effected by the poison and the defective oxygenation—hence lassitude and weariness, disturbed functions, or congestion of some or all of the viscera, and a lower temperature.”¹

To these effects may be added prostration of the entire constitution and disturbance of the circulation; while the diminution of the fibrin explains the want of cohesion in the solids, the ecchymosis on the skin and in the cellular tissues, the bleeding from the nose, gums,² stomach, bowels, and other outlets, the black vomit, &c.

CHAPTER XIX.

CRITICAL DAYS AND CRITICAL EFFORTS.

It is very generally admitted by writers on the yellow fever, of both tropical and temperate regions, that the influence of critical days, in regard as well to a favourable or unfavourable issue of the case, seldom fails to be appreciable in this disease. That this has been denied by respectable authorities, is doubtless true. We are told by some writers, that the periods at which the amaril typhus, under which denomination is described the yellow fever of temperate climates, terminates, or is judged (*se juge*), are undetermined (*n'ont rien de fix*); that, hence, it is in no way possible to foresee the nature of its issue, by the supputation of certain determinate days; and that this can only be done by taking a general survey of the time which has already elapsed since the commencement of the attack, and endeavouring to foresee, from a consideration of the progress, character, and intensity of the symptoms, that which will be required before the disease comes to an issue.³ “Hence, it is not

¹ Brit. and For. Med.-Chir. Rev., iii. 95-6.

² Dublin J., vii. 219.

³ Rochoux, p. 519.

as being even or uneven, or as forming part of any number, that the influence of days must be appreciated, but only as constituting portions of time, and so far as they recede from the period of the invasion."¹

All this may have been the case at Barcelona in 1822, where Dr. Rochoux had an opportunity of observing the yellow fever of temperate climates. Or, rather, all this may be true, so far as relates to the cases which fell under his notice; but, while admitting this, and also that it is impossible at times—not in one section of country only, but everywhere—to mark any particular critical days—death and recovery happening irregularly without any evident regard to special periods; and though such irregularities may occur very frequently during certain seasons, and especially in certain forms of the disease, the experience obtained in this country is in direct opposition to the idea of extending the statement to the fever of temperate regions generally; while facts in abundance show that in tropical climates the disease is, as M. Rochoux himself admits, under the influence of critical days. Nor could it be difficult to show that the same influence is attested by writers on the fever of Spain and Italy. Dr. Rush, in his account of the epidemic of 1793, says: "The termination of this fever in life and death was much more frequent on the third, fifth, seventh, ninth, and eleventh days, than is common in the mild remittent fever. When death occurred on the even days, it seemed to be the effect of a violent paroxysm of fever, or of great vigor of constitution, or of the force of medicine, which protracted some of the motions of life beyond the close of the odd days which have been mentioned" (iii. 73). In 1794, as he informs us elsewhere, "the crisis of the fever occurred on uneven days more frequently than in the year 1793;" but he adds that critical days "were observable in almost every case in which the disease was not strangled in its birth" (iii. 214–15)—applying, approvingly, to this fever what Chisholm says of the fever of Grenada—that he had not met with any disease in which the periods were more accurately ascertained. Dr. Rush had noted the same fact in relation to the fever of 1762 (iii. 44). This regularity was noticed by Dr. Currie, who remarks that, when the disease terminated favourably, it was mostly on the third, fifth, or seventh day—when the reverse on the fourth, sixth, or eighth day.² A similar regularity in the observance of critical days is pointed out by Nassy (p. 24), Dalmas (p. 232), Baxter (xxi. 3), Merrill (ix. 246), Lining (ii. 416–17), and other writers of our country, and may be inferred from the reference made by a greater number of authorities to the particular periods at which the disease terminates. "It was evident," says Dr. Tully, "that this fever, when regular, is a disease of seven days, though the severity of the symptoms was often such, on the one hand, as to terminate the life of the patient on the fifth, or even the third; or, in some cases, when recovery took place, it was, on the other hand, sometimes protracted to the fourteenth day" (p. 301).

In the fever of Seville in 1819, Velasquez found that patients commonly

¹ Rochoux, p. 520.

² Fever of 1797, p. 220.

recovered on the third, fifth, or seventh day, if the happy efforts of nature, or the well-directed succours of art, excited favourable evacuations (p. 10). Further on he says, that whatever be the precise form of movement which characterizes the febrile course, the third day and the fifth are days conspicuous for changes which indicate final results. On these days, the third in some, the fifth in others, changes manifest themselves which are regarded throughout as diagnostic of the disease, and generally prognostic of the issue. In speaking of the fever of Xeres and Cadiz of the next year, Dr. R. Jackson is still more explicit: "The fatal changes happen at common critical periods; and when stagnant life resumes a course which leads to health, it is always at one or other of the known critical days, viz: third, fifth, or seventh, that the commencement of the favourable change occurs."¹ Were it necessary to prolong the investigation, it would be easy to show, by a reference to other writers,² that the epidemics of Cadiz in 1800 and 1819, of Malaga in 1804, of Gibraltar in 1828, and even of Barcelona in 1821, could be adduced in favour of the same phenomena.

If, in turning now our inquiries to the fever of tropical climates, we find that Lind (p. 285), Hunter (p. 87), Bally (p. 272), Savarésy (p. 290), Blicke (p. 205), Fontana (p. 73), deny the influence of critical days, or the disposition to a change as regards the termination of the disease at determinate periods; if Dr. H. McLean noticed the impossibility of marking critical days in the fever of St. Domingo, from the irregularity of the periods at which death or recoveries happened; if De Madrid could say, as the result of his experience: "El sudor y los deposiciones que alivian, anuncian que los sólidos van recobrando su accion, y son por tanto favorables; pero no hay crisis en esta enfermedad. El alivio no es efecto de las deposiciones, sino de la reaccion de los organos;"³ others, perhaps the greater number of writers, have come to an opposite conclusion;⁴ some with regard to the periods at which salutary changes take place—others with reference to those at which death occurs; and even Dr. McLean acknowledges that the fifth day, the seventh, and the eleventh, appeared in some degree critical, though, he adds, not by any means in a certain invariable order (p. 93).

"It is a general observation," says Chisholm (i. 192), "that, in malignant fevers, the critical periods are more distinctly marked than in any other; the disease before us afforded an additional proof of this. I have not met with any disease in the West Indies in which these periods were not distinctly ascertained. The disappearance of the disease, or the death of the patient, always happened on odd days; but the change in the state of the disease which preceded either event, took place on even days. Thus, if the patient

¹ Fever of Spain, pp. 87, 110.

² Caisergues, p. 173; Berthe, p. 102; Arejula, p. 448; Amiel, xxxv. 279; Ib. in Johnson, p. 267; Pariset, Obs., p. 31; Audouard, pp. 59, 66; O'Halloran, p. 84.

³ Part ii. 34.

⁴ Desperrière, F. de St. Domingue, p. 119; Desportes, Mal. de St. Domingue, i. 198; Dazille, Mal. des Nègres, pp. 36, 65; Bajon, Mém. sur Cayenne, i., Mém. ii; Leblond, Fièvre Jaune, p. 43; Levacher, Guide Méd., p. 50; Evans, p. 250; Caillot, p. 159; Leprieux, ii. 88; J. Clark, p. 16; Dariste, p. 138; Gilbert, p. 78.

was worse on the evening of the second day, he would die on the third; if worse on the fourth, he would die on the fifth; and so on as far as the fourteenth day. Beyond that period, I have not seen an instance of the disease ending fatally, although it has been protracted, in a few instances, to the twenty-first day. In the same manner, if the patient felt better, or if there was an evident abatement of the symptoms on the second, fourth, sixth, &c., days, the revolutions of the disease would happen on the following days."

Rochoux, who, as we have seen, denies the influence of critical days in the yellow fever of temperate climates, is no less positive than Chisholm as to their influence within the tropics, where he had ample means of investigating the disease. After remarking that experience confirms daily the observation of the English physician, he says: "In effect, more than one-half of the subjects affected with the yellow fever, perish from the fourth to the fifth day. Next come the seventh, the ninth, and perhaps the eleventh day, beyond which I have not seen the disease extend. The number of terminations diminishes in proportion as these extend beyond the fifth day. It is not to be inferred from this that persons do not die or recover on the other days. Every one knows, that in violent epidemics, some are found to die before the completion of the third day. Some die and some recover also on the fourth, sixth, eighth and tenth days; but, in general, these last days are those in which we observe the changes indicative of the manner in which the disease will terminate on one of the following odd days. This knowledge of the influence of critical signs, is in some sort general (*vulgaire*) in the colony (Guadaloupe). People inquire as to the condition of patients for whom they feel an interest, on the fourth or fifth day; and the prognostics which individuals, strangers to the art, draw from that single indication, exhibit a degree of certainty which a physician, accustomed in France to find the progress of disease much less under the control of critical days, would feel disposed to call into doubt" (pp. 347, 8).

The readers of Dr. R. Jackson, one of the highest authorities on the subject of tropical fevers, need scarcely be told that he was a strong advocate of the influence of critical days, and will recollect how forcibly he illustrated the fact that the changes or terminations in all cases are under the influence of a law in the system which acts periodically—though in a manner we do not comprehend. He remarks that if an impression be made upon the system by the action of a morbid cause, and if the forward course of that action be arrested by the application of strong measures, the effect of the original impression, notwithstanding this forcible effect, still remains, so as to impede the healthy action of the system in a greater or less degree, until perfect solution takes place under the influence of a known critical period. For example, if a febrile disease of the major degree of violence be arrested, the progressive course checked by abstraction of blood or other means, the disease may be said to cease, but the action of health is rarely effective in all the functions of the system before the third, fifth, and sometimes the seventh day from the date of the first attack. These are known critical days; and it is at these periods principally that changes are observed to take place in the

form and character of febrile diseases, whether partial or general, temporary or permanent; they are observed independently of the operation of medical means; if art have checked the course, the influence of the critical period accomplishes or completes the cure.¹ In another work, Dr. Jackson says: "The laws of critical days are regular and undeviating, though the crisis be not always perfect and complete. This is said of the different forms of endemic fever, even of the most concentrated which constitute the genuine yellow fever of authors." (*Outline*, p. 243.)

Much may be said concerning the above law relative to the period of seven and intermediate critical days in fevers; for it is one which, as Dr. Laycock has forcibly shown, is of very general application in the explanation of vital phenomena in health and disease. Everything, indeed, leads to the conclusion that a period of seven days, and definite fractions or multiples of that period, are very prominent in the series of phenomena called critical days, and the operations of the system generally. It forms part of the great fact of vital periodicity, to which attention was called by some of the most ancient writers who have left records of their observations. The doctrine of septenaries is literally as old as the hills. Originating probably with the Chaldeans or Egyptians, it formed part of the system of Pythagoras; and its application, to the phenomena of disease particularly, is discussed by Hippocrates, Diocles, Galen, Celsus, and others. In his ingenious speculations on the periodicity of the phenomena of life, Dr. Laycock has shown that the critical days of health and the critical days of fevers produced by the entrance of a poison into the system—whether that poison consist in malaria, or in a contagious matter—are identical; that the depression of the system will, in consequence, take place at those critical days, and that a fever depending on a poison is more likely to appear on those days than on any other. In confirmation of this, he states that the latent period in most diseases is regulated by weeks, as is also the latent period of animal poisons. He remarks that, in accordance with this law, the latent period of fever rarely extends beyond twenty-eight days. "If we take menstruation as a type of the critical days, and suppose that a movement takes place every seven days, gradually becoming more intense at each up to the fourth week, we have fever days, at least in every month in which the peculiar symptoms of the poison, whether malarious, exanthematous, or contagious, may exhibit themselves; probably the number may be greater; but if one or two of these days be passed over without an outburst of febrile action, it is scarcely possible that the third or fourth will."²

In the fevers of hot climates, which are all, or for the most part, malarial, the doctrine of critical days, such as it has been transmitted to us from the days of Hippocrates, has been amply verified. On this subject, we have the testimony of high professional authorities. In the fevers of Europe, it has been found equally applicable;³ and, in this country, when the efforts of

¹ Sketch, i. 205-6.

² London Lancet, Oct. 1842, p. 161.

³ Hildenbrandt, *Med. Pract.*, pt. i. chap. 5; De Haen, *Pract. Med.*, pt. i. cap. 4; Baglivi, *Opera*, p. 80.

nature are not too much interfered with by an *heroic*, perturbative, and so called abortive treatment, the Hippocratic doctrine often shines out in all its purity. To this fact, which some twenty or thirty years ago it would have been considered heresy to allude to approvingly, the eyes of our more enlightened physicians are opening. Even in sections of country where formerly the idea of critical days met with most opposition, a very different sentiment is now found to prevail in the minds of correct, careful, and enlightened observers. Let one example suffice. Dr. Anderson, of Alabama, in a prize essay of merit, after remarking that a second exacerbation takes place the day following an attack of fever, generally after meridian, adds: "This, in turn, gives way during the night, or towards morning, and another remission, more or less decided, is observed. If the fever take the double tertian type, the next exacerbation will come on during the morning, and will be followed by another the succeeding evening. The fever thus continues, sometimes rising during the same hour every day, and sometimes later on alternate days, until the sixth, when, if the observations of the writer of this essay are worth anything, the fever (if it terminate favourably) has a decided spontaneous tendency to decline, and leaves the patient free from disease on the seventh day." Again: "The fever (malarial) is generally at its height on the fifth day; and, in bad cases, this is the day of danger. In malignant tertian intermittents, the fifth is the day for the third paroxysm, universally known to be the most hazardous; and, when death occurs in the disease, it commonly takes place either on that day or during the next." "Commencing practice," continues Dr. Anderson—whose fate, in that respect, has been similar to that of hundreds of physicians of this country—"with an utter disbelief in the doctrine of critical days, the contrary opinion has been forced upon me by actual observation. I am aware that the idea is discarded by a majority of physicians of the day; but I think that if any practitioner in the Southwest will carefully note down the days in which malarious fever makes its appearance, and record accurately its subsequent course, he will find that the doctrine has a foundation in fact, and is worthy of some consideration. It is true that the fever may often be made much lighter, or apparently arrested by the administration of quinia during the remissions; but it will generally be observed that the patient does not frankly recover, and that it is not until the seventh day that he seems actually well, and clear of all symptoms of the disease."¹ This, as may be perceived, is in exact accordance with the remarks of Dr. Jackson relative to the yellow fever of the West Indies, and with what may be seen everywhere. The fever may be greatly modified or moderated by therapeutic means; but perfect recovery is not obtained. Something remains behind, which will not be got rid of except on one of those days set apart for that purpose by nature.

But, however well founded may be the influence exercised by critical periods on the solution of the disease; it is not the less certain that, as a gene-

¹ Proceedings of the Med. Assoc. of Alabama for 1852, p. 107.

ral rule, the yellow fever may be classed among those diseases which frequently come to a close—in recovery or death—without the occurrence of any of those commotions in the system which take place at the regular periods above alluded to, and which are viewed in the light of critical efforts on the part of the system to rid itself of the morbid cause under which it labours. The symptoms, in most instances, gradually abate, or become aggravated, until, at last, the disease ends in recovery or death, at the stated periods in question. Such has been often the case in this city¹ and country. “The fever accompanied—as Dr. Lining long ago remarked—with those symptoms, terminated on the third day, or generally in less than seventy-two hours from the first attack, not by any assimilation or coction and excretion of the morbid matter; for, if by the latter, there would have been some critical discharge by sweat, urine, stool, or other ways, none of which happened; and if by the former, nothing then would have remained but great debility. No, this fever did not terminate in either of these salutary ways, except in some, who were happy enough to have the disease conquered in the beginning by proper evacuations, and by keeping up a plentiful sweat, till the total solution of the fever, by proper mild diaphoretics and diluents.” Such has also been found to be the case in the South of Europe, and, time after time, in tropical countries.²

True as this may be, however, it is not the less true that, in all places and during all epidemics cases not unfrequently occur, marked in their termination by a natural effort, of a more or less decided character, through some one or other of the parts usually implicated in critical commotions—the skin, alimentary canal, urinary organs, capillary system, &c. In this city, as elsewhere, such efforts have been noticed; and though, from the perturbing nature of the mode of treatment usually pursued, it is sometimes difficult to ascertain, when the disease is relieved after alvine evacuation, how far the result is to be ascribed to the effort of nature, and how far to the medicine used, yet the share of the former may generally be made out from the quality of the evacuations, the greater facility with which they can be obtained, and the rapidity of the relief which follows their appearance. Be this as it may, the occurrence of critical efforts by alvine discharges has been observed among us, even in the hands of the most energetic administrators of purgatives, though more frequently by those who are sparing in the use of such medicines. They are recorded everywhere else. They are mentioned, in more or less explicit terms, whether as the result of spontaneous efforts, or as aided by art, by the highest authorities in this and other countries.³

¹ Deveze, pp. 31, 45, 46; Nassy, p. 24; Lining, ii. 416, 417; A. Smith, xxv. 501; Gros, p. 14; Girardin, pp. 35, 42.

² Pariset, p. 437; Bally, pp. 267, 272; Dubreuil, viii. 334; Gilbert, p. 78; Dariste, p. 135; Lempriere, i. 88; H. McLean, p. 95; Fontana, p. 73; Savarésy, pp. 290–91; Jackson, Tr., p. 263; Madrid, pt. ii. p. 34; Blicke, p. 205; Mabit, p. 12; Dupont, p. 21; Rayet, p. 21; Amiel, Edin. Journ., xxxv. 279.

³ Rush, iv. 32, 33; Monges, ii. 58; Currie, Fev. of 1793, pp. 23, 35, 36; Ib., Fev. of 1797, p. 219; Cathrall, p. 36; Deveze, pp. 31, 36; Palloni, p. 6; Caisergues, pp. 123,

Critical efforts through the urinary organs, as manifested by increased discharge or a turbid sedimentous condition of the fluid, have not less frequently been noticed; for though, in this city, they have not always proved as complete and effective as the preceding, yet they have done so in a sufficient manner to impart to the discharge the character of a truly critical manifestation. They are mentioned in that connection by Deveze (pp. 31, 46), Currie (37), and others; and, so far as relates to the fever of other places, may be said to have been a subject of general observation.¹

Critical efforts by sweat have been perhaps more frequently noticed, and have undoubtedly proved more complete and effectual.²

Epistaxis has frequently appeared, here and elsewhere, to assume the character of a salutary critical discharge, especially when it occurs early, and at the close of the period of irritation.³ Menorrhagia, or the menstrual discharge,⁴ as well, indeed, as other hemorrhages—of the anus, mouth, bladder, &c., have often produced the same salutary effects, and, in consequence been viewed in the same light.⁵ On this subject, Mr. Catel remarks: “It is usually from the third to the fifth day that the worst symptoms present themselves. One would almost suppose that nature, reacting violently against the inflam-

173; Berthe, p. 123; Audouard, p. 59; Blin, p. 11; O'Halloran, p. 130; Merrill, ix. 247; Dalmas, p. 233; C. Drake, xxi. 135; Valentin, p. 175; Gros, p. 17; Moultrie, p. 16; Savarésy, p. 286; Lind, p. 285; Desportes, i. 196, 197; Jackson, Sketch, i. 160, 173; Blane, pp. 412–414; Fontana, p. 73; Maher, pp. 843, 893; Osgood, pp. 14, 15; Gillespie, p. 40; Lempriere, ii. 89; Desperrière, p. 71; Moseley, pp. 448, 449, 468, 469; Towne, p. 22; Gilbert, pp. 73–78; H. McLean, p. 95; Caillot, p. 158; Bally, pp. 473, 474; Pugnet, p. 356; Rochoux, pp. 345, 521, 549; Joubert, p. 966.

¹ Townsend, p. 155; Palloni, p. 6; *Ib.*, *Edinb. Journ.*, ii. 85; Berthe, p. 123; Caisergues, pp. 50, 51, 173; Maher, p. 843, 893; Desportes, i. 197; Towne, p. 22; Savarésy, p. 286; Gilbert, p. 78; Jackson, p. 173; Blane, pp. 412, 414; Pugnet, pp. 356, 360; Osgood, p. 15; Moseley, p. 449; Caillot, p. 158; Bally, pp. 473, 474; Rochoux, pp. 345, 521, 549; Jourdain, v. 259, 260; O'Halloran, p. 130; Valentin, p. 175.

² Rush, iii. 63; Monges, ii. 58; *New Orleans in 1839*, p. 338; Moultrie, p. 16; Drysdale, i. 42; Merrill, ix. 247; Dalmas, p. 23; Hill, v. 90; Palloni, p. 6; *Ib.*, *Edin. Journ.*, ii. 85; Amiel, pp. 167, 168; Arejula, *Edinb. Journ.*, i. 449; Caisergues, pp. 173–180; Jourdain, v. 259; Audouard, p. 66; Pariset, *Obs.*, pp. 10, 14, 31; *Ib.*, *Fev. of Barcelona*, p. 394; Blin, p. 11; Berthe, p. 124; O'Halloran, p. 130; Arnold, p. 17; Lind, p. 285; Maher, p. 893; Pugnet, p. 360; Savarésy, p. 286–288; Desportes, i. 197; Fontana, p. 74; Jackson, i. 160, 186; Desperrière, p. 71; Gillespie, pp. 45, 56, 57; Imray, liii. 85; McArthur, p. 353; McLean, p. 95; Caillot, p. 158; Bally, pp. 473, 474; Rochoux, pp. 345, 521, 549; Hume, p. 216.

³ Sam. Jackson, p. 60; Currie, p. 35; Rush, iii. 151; Cathrall, p. 36; Hogg, *Western Journ.*, i. 412–414; Pariset, *Obs.*, p. 31; Velasquez, p. 14; Desperrière, pp. 71, 126; Gilbert, p. 79; J. Clark, p. 7; Dariste, p. 177; Bally, p. 276; H. McLean, p. 95; Rochoux, p. 331; Desportes, i. 198; O'Halloran, p. 130; Moseley, p. 448; Gros, p. 17; Maher, p. 893.

⁴ Dariste, p. 177; Velasquez, p. 14; Pariset, *Obs.*, p. 31; Deveze, p. 31; Lining, ii. 431; O'Halloran, p. 130.

⁵ Deveze, p. 46; Fellowes, p. 62; Audouard, p. 68; Caillot, p. 158; Maher, 854, 880; Rochoux, p. 345; Desportes, i. 198; O'Halloran, p. 130; Catel, *Ann. Marit.*, p. 226; Fenner (1853), p. 50.

mation which threatens to disorganize the viscera, endeavours to destroy, through means of hemorrhages, the evil which oppresses her. These hemorrhages are active, and so much the more abundant, as the patient has lost less blood by venesection. If we are to judge by the result of observation and experience, we should say that these hemorrhages are critical, and consequently salutary, whenever they flow externally. We have seen individuals lose as much as three litres (or quarts) of blood by exudation from the tongue alone, and four litres by the nose. These individuals have recovered. Even hemorrhages by the stomach are not fatal, for we have seen several cases of recovery after black vomit." These statements are rather startling; but they are useful in showing the critical character of hemorrhages, and the quantity of blood that may be lost without danger to life. In 1853, at New Orleans, hemorrhage was not always an unfavourable symptom. "On the contrary," says Dr. Fenner, "it often indicated the approach of a *salutary crisis*. Indeed, it is the natural crisis of this type of fever, when its course is not interrupted by art. How often have I hailed with joy the appearance of a moderate hemorrhage from some *safe part*—as the nose, gums, or uterus—in the critical stage of yellow fever, when it had been previously altogether uncertain how the case was going to terminate. At this stage of the disease, a small quantity of blood flowing spontaneously from any part, except the stomach, is most generally followed by the happiest effects, reducing, nay, removing febrile excitement, and allaying nervous inquietude."¹ Critical efforts occasionally manifest themselves by spontaneous salivation,² by vomiting,³ swelling of the parotid gland,⁴ by bubos,⁵ by anthrax and carbuncles,⁶ cutaneous eruptions about the mouth and other parts,⁷ abscesses,⁸ local inflammations of small joints,⁹ partial gangrene of the skin, serotum, &c.,¹⁰ local inflammations of an erysipelatous character,¹¹ and furuncles or boils.¹²

More than a century ago, Dr. Towne, of Barbadoes, called attention to the constant tendency nature shows to discharge herself of the burden she labours under, by dispersing and throwing out of the mass of blood that load of bile which, as he thought, is the principal agent in this important mischief; and

¹ Fenner, p. 50.

² Deveze, p. 46; Makittrick, p. 70.

³ Desportes, i. 198; Deveze, p. 46.

⁴ Deveze, p. 32; Desportes, i. 198; Rochoux, p. 346; Pugnet, p. 360; Lind, p. 285; Rush, iii. 67, 68; Lefort, p. 561; Catel, Ann. Marit. (1844), iv. 227; Chisholm, i. 178, &c.

⁵ Dict. des Sci. Méd., xv. 338; Chisholm, p. 146, 1st ed.; Lind, p. 285.

⁶ Chisholm, p. 146, 1st ed., i. 179, 2d ed.; Desportes, i. 197, 199, 214, 223; Rush, iii. 69; A. Hosack, p. 19; Finlay, p. 22.

⁷ Cathrall, p. 36; Rush, iii. 44; Moultrie, p. 16; Drysdale, i. 131; Warren, p. 17; Lind, p. 285; Arejula, p. 183; Finlay, p. 22; Gillkrest, ii. 274; Gilbert, p. 78; Rouppe, p. 307; Desportes, i. 197, 198; Pugnet, p. 360; Pariset, p. 434; R. Jackson, i. 76, 187; Imray, liii. 85; Arnold, p. 19; Blane, p. 439; Chisholm, i. 179; Fellowes, pp. 57, 78, &c.

⁸ Cathrall, p. 39; Pariset, p. 434; Bally, p. 253.

⁹ H. McLean, p. 95.

¹⁰ Deveze, p. 46; Laso, p. 250; Chisholm, p. 122, 1st ed.; Lefort, p. 561; Maher, p. 893.

¹¹ Lefort, p. 561.

¹² Warren, p. 17; Desportes, i. 197, 198; A. Hosack, p. 19; Moseley, p. 449; Fontana, pp. 73, 74; Hunter, p. 77.

affirmed that he had very rarely known a happy *crisis* obtained where it had not plainly appeared to be the consequence of nature having succeeded in this attempt.

According to this view of the subject, the regular *crisis* of this fever generally discovers itself by a suffusion of bile all over the surface of the body about the third day. "The saffron tincture is frequently observed in the space of twelve hours after the attack, if you carefully inspect the coats of the eyes; and the sooner it appears, the more encouraging is the *prognostic*, if the intention of nature be not perverted by the preposterous use of cordials and alexipharmics. If blisters are applied, even in this early stage of the distemper (which the vehemence of the case often requires), they will make a plentiful discharge of a bilious serum; the urine will likewise be of the same colour, as well as the intestinal evacuations, which, if necessary, may easily be procured by manna, cream of tartar, and such like laxative medicines."¹

The opinions of Towne were soon after disputed by Warren, who, regarding jaundice as the result of a more complete colliquation or dissolution of the red globules of the blood into a yellowish serum, which naturally soon gives that tincture to the whole skin, could not view it as a salutary crisis. Warren claims the credit of having convinced Towne of these mistakes, and goes so far as to add that he must do him the justice to declare that he frankly retracted his opinion before he died, and would willingly have called in the copies of his book, could he have found means of doing it.²

Hillary, in like manner, opposed the opinion, and stated that this total yellowness, so far from being always critical and an encouraging prognostic, most commonly, on the contrary, proves a mortal symptom; remarking, with Warren, that, when it comes on soon, it shows a greater colliquation and dissolution of the blood, and a gangrenescent state of this fluid (p. 149).

Moseley, who came not long after, foresaw plainly that both these opposite opinions were erroneous, but only in being each of them carried too far—truth lying between them. "A yellow suffusion may," he says, "be either critical or symptomatical: critical, as Towne supposes—but it must be when there is a tranquil cessation, without languor, of all the other symptoms, with warm perspiration; and symptomatical, as Hillary supposes, when accompanied with lassitude, nausea, vomiting, colliquative sweats, and sunk pulse. The case, in my opinion," Moseley continues, "stands exactly thus; notwithstanding Hillary's idea that the yellowness cannot be critical, should it appear before the eighth or ninth day." "But Hillary must often have had opportunities, which perhaps he had forgotten, to know that his contradiction of Towne was ill-founded" (pp. 468, 469).

Hillary, indeed, seems to acknowledge it, for he grants "that this yellow suffusion of bile upon the surface of the body has, at a chance time, though very seldom, proved critical; but then it did not come on till the eighth or ninth day, nor appear till the coma and all the other bad symptoms began to abate: and, as the yellowness increases, they all decrease; but this very rarely

¹ Towne, pp. 23, 24.

² Warren, pp. 13–15.

happens." Most commonly it is the reverse, especially when it comes on soon, as it is then not only symptomatic, but ushers in the last and fatal symptoms. "So that, from the first appearance of this symptomatic yellowness, we may say the patient is in the last stage of the disease, how soon soever it may come on; though, in some, it has not come on till the eighth or ninth day, and then is usually critical—but this very rarely happens" (pp. 149, 150).

Among more modern observers, the idea of placing jaundice among the critical changes—for good or for evil—is not uncommon;¹ but, as may be presumed from what was said in preceding chapters, the effort is never salutary except when it occurs at a late period. It is difficult to resist a suspicion as to the correctness of the observation that would ascribe an efficient agency to some of the phenomena mentioned above. We may unite in opinion with those writers who think that it has happened in more instances than one that the desire to trace every change which takes place in the course of the disease, and is followed by a restoration to health, to a critical effort of nature, has led physicians to regard in that light phenomena of a fortuitous character—mere coincidences. There is reason to believe, indeed, that the name has been given occasionally to pathological modifications which have had rather the effect of retarding than hastening the cure; in other instances to some which, arising from various causes, have done good on the principle of revulsion; or again, to some which, happening at the time the disease was taking a favourable turn, exercised no influence on the nature of the results, and may, perhaps, be properly regarded as the effect, not the cause of returning health.

CHAPTER XX.

TYPE OF THE YELLOW FEVER.

As may be perceived from the account I have given of the symptoms and course of the yellow fever as it has appeared in this city, a febrile stage of about seventy hours' duration, more or less, is succeeded by a period of calm, or complete cessation of fever. This, in some cases, constitutes the harbinger of a prompt recovery; while, in other instances, it is, after the lapse of from twelve to thirty-six hours—sometimes of a day or two—followed by the more alarming and malignant symptoms which, after some time, gradually abate and terminate in health; or, as more frequently occurs, assume a more serious aspect, and lead to a fatal termination. This state of remission—the *stadium* of Lining, and the *metaptosis* of Moseley (during which the pulse returns to its natural standard, the tongue, skin, and other surfaces lose their morbid appearances, and most of the other symptoms disappear completely,

¹ Catel, Arejula, Fellowes, Gilbert.

or in great part) occurs at the period mentioned, with greater or less abruptness. It is never, or very seldom, followed by a recurrence of febrile excitement, and is observed in the large majority of cases, indeed in all, except the milder forms of the disease, in which it is either less marked or entirely absent—the febrile excitement in these continuing beyond the period mentioned, and gradually dying away, or being carried off through the agency of some critical effort.

A reference to the writings of Rush,¹ Currie,² Caldwell,³ Barnwell (p. 373), Monges (ii. 56), Cathrall (pp. 6, 30), S. Jackson (p. 56), will show that, in this city, the fever has, at all its epidemic recurrences, exhibited this peculiarity. In every other place throughout this country, from Galveston to Boston, a similar phenomenon has been observed, thereby fully justifying the remark of the late Dr. Miller, of New York, that “if any symptom can be said to distinguish the yellow fever from other forms of pestilence, this (the metaptosis) deserves, perhaps, to be selected.”⁴ Hence, we find it dwelt upon by every writer on the yellow fever of America from the days of Moultrie to our own.⁵ As regards the yellow fever of the West Indies, the coast of Africa, or the coast of Mexico and South America, some writers—and among them Dr. Rochoux, Dr. Hastings, Dr. Boyd, &c.—have either taken no particular notice of the peculiarly sudden cessation and total disappearance of febrile excitement, or mentioned the abrupt occurrence of an apyretic state after a high excitement of from one to four days’ continuance, as a phenomenon rarely encountered. But whatever may be the result of their experience on the subject, and even admitting that the description they give of the disease is applicable to true yellow fever, certain it is, that other and very many writers on the fever of those regions, have represented it as pursuing a course, in respect to the phenomenon in question, precisely similar to that to which I have alluded. Thus Dr. Bancroft, after speaking of a paroxysm of thirty-six to seventy-two hours, says: “A remission then occurs, in which many of the symptoms subside, so as often to induce a belief that the fever is at an end, and recovery about to take place. Frequently, however, the foundation of irreparable injury to the brain or stomach has already been laid in the former paroxysm, and in such cases the remission is short and imperfect. During these remissions the pulse often returns apparently to the condition of health, the skin feels cool and moist; and the intellect, if previously disturbed, sometimes becomes clear. After a certain interval, this remitting stage is succeeded by another, which may be called the second paroxysm” (p. 12). I need scarcely point out the similarity which is therein shown to exist, on the point in question, between our yellow fever and that to which Dr. Ban-

¹ Fev. of 1793, iii. 73; 1797, iv. 13.

² Fev. of 1793, pp. 23, 25.

³ Mem. (1826), p. 104; Fever of 1805, p. 84.

⁴ Obs. on Y. F., Wks., p. 56.

⁵ Moultrie, p. 4; Lining, ii. 416; Shecut, p. 119; Addoms, p. 10; Waring, p. 45; J. Warren, in Tytler, p. 502; Seaman, pp. 8, 9; Tully, pp. 297–8; Townsend, p. 179; Ticknor, iii. 275; Dalmas, p. 11; Girardin, p. 35; Thomas, p. 83; Drysdale, i. 41; Cartwright, ix. 14; Dickson, iii. 255; Ib., Essays, i. 349; Revere, iii. 225; Stone, vi. 554; Lewis, N. O. J., i. 298; Wragg, x. 70; Dupuy de Chambery, xxi. 17; A. Smith, xxv. 561; N. O. 1819, p. 8; N. O. 1820, p. 9; N. O. 1839, p. 326.

croft has reference; and the reader who is interested in such matters will find that the statement made by that distinguished physician accords with that of almost every other observer in the West Indies, the coast of Africa, and other parts of tropical regions; many of whom, indeed, have been even more explicit than Dr. Baneroff in reference to the remission in question.¹ Nor is it less certain that, with the exception of Louis, and a few more of less note, who, as stated in reference to Rochoux and others, take no particular notice of the peculiarity in question, every writer on the yellow fever of Southern Europe, even those who have described the very epidemic observed by the above-mentioned physicians, have dwelt upon it with more or less emphasis. Such has been the case as regards the fever of Leghorn in 1804 (Dufour, iv. 51; Palloni, p. 4); of Cadiz, in various years (Caisergues, pp. 168, 177; Berthe, p. 70; Arejula, p. 161; Pariset, *Obs.*, p. 30; Blin, p. 6; R. Jackson, pp. 74, 111; O'Halloran, p. 80); of Barcelona (Pariset, pp. 380, 397; Rochoux, pp. 117, 516); of Gibraltar (Gilpin, v. 323; Pym, xxxv. 41; Amiel, in *Johnson*, p. 264); of Minorea (Boyd, p. 300); of Seville (Velasquez, in *Pariset*, p. 10); of Port du Passage (Jourdain, v. 260); and, in fact, of every place where the fever has appeared on that side of the Atlantic.

This remission, or rather sudden cessation of fever after one long paroxysm—the only one which occurs—may be regarded, therefore, in the light of a well ascertained fact. It is very seldom, if ever, succeeded by symptoms of vascular reaction, but by an apyretic condition, during which, in severe and aggravated cases, the more malignant phenomena present themselves. It constitutes a marked and distinctive characteristic of yellow fever, and is pointed out as such by every writer, and in every clime. As already stated, it seldom fails to present itself; the exceptions to the rule are rare, and occur in mild cases in which symptoms of the first stage alone, more or less prolonged, are noticed, or in the violent and congestive forms of the disease, when those of the last stage are not preceded by a period of reaction; while, on the other hand, it is never observed in other forms of febrile complaints—the oriental plague, perhaps, excepted. So true, indeed, is this, that the occurrence in question is of itself almost sufficient to enable us to diagnose the case, while its absence may, unless under the circumstances mentioned, justify a doubt as to the genuineness of the disease, or lead us either to view it as the offspring of a different poison, or to suspect the existence of a complication.²

The yellow fever may, therefore, strictly speaking, be viewed as a disease of a single regular paroxysm. Nothing bearing any analogy to such a

¹ Warren, p. 10; Makittrick, p. 71; Dancer, p. 83; Gilbert, p. 65; Caillot, p. 18; Maher, p. 849; Joubert, p. 967; Bally, p. 208; H. McLean, p. 72; J. Clark, p. 11; Furlong, p. 290; Levacher, p. 73; Doughty, p. 61; Blair, p. 64; Dariste, p. 160; Dyott, p. 1003; Curtin, *Med. Com.*, ix. 237; Wilson, p. 178; Belcher, xxiii. 250; W. Barry, in Boyle, p. 271; Imray, pp. liii. 80, lxiv. 336; Dickinson, p. 32; Heastie, p. 19; Moseley, pp. 436–7; Ruz, iii. 55; R. Jackson, i. 71; Tr., p. 260; Lempriere, ii. 61; Osgood, p. 11; Chisholm, i. 147, 150; Grant, p. 31; Gillespie, p. 143; Frost, xiii. 30, 34; Fontana, p. 73; Madrid, p. 25; Holliday, p. 12; Carter, p. 5; Lallemant, p. 51.

² Lewis, N. O. J., i. 428.

paroxysm recurs after the cessation of the period of calm in question. Febrile excitement may, and doubtless does, sometimes occur. But I repeat, after the accession of the metaptosis, fever, as a distinct feature of the disease, ceases; and we may agree with those who affirm that what has been taken for a return of the paroxysm after the remission is, as many good observers have long before remarked, an invasion of fever of uncertain type, varying according to the nature of the injury inflicted. It must be viewed in the light of a fever of irritation—the effect of some complicating ailment—which may or may not exist, without impairing, by its presence or absence, the purity of the disease of which it does not constitute an integral part.

As to the type of that paroxysm—whether, during its continuance, the fever observes a continuous course, or whether it presents alternations of exacerbation and diminution which would entitle it to the appellation of a paroxysmal disease of a remittent or intermittent character—a great difference of opinion has long existed, and continues to exist.

Dr. Rush, in the several accounts he has left of the epidemics of this city, while allowing that the disease was sometimes of a continued type, speaks of the remittent as being very common. Of the fever of 1793, he says: “The remissions were more frequent in this than in the common bilious fever. They occurred in the forenoon” (iii. 73). In regard to the epidemic of 1794, he makes similar statements (iii. 213, 214). So, also, in regard to that of 1797; adding, however, that, on the latter occasion, it frequently assumed the character which is given of the same fever in Charleston, by Dr. Lining. It came on without chill, and continued, without any remission, for three days, after which the patient believed himself well (iv. 13).

Deveze states that the fever assumes often the continued type, but is more frequently remittent (pp. 34, 35). In the fever of 1820, remissions—in milder cases especially—were noticed by Dr. Jackson. Similar statements have been made, or the occurrence has been hinted at, by many other writers, both in this and other cities of this country.¹

In Spain and Italy,² as likewise in tropical regions,³ the remittent cha-

¹ Barnwell, p. 373; Valentin, pp. 172, 173; Archer, p. 66; Hill, p. 90; Drysdale, i. 41; Toulmin (Mobile), p. 197; Revere, iii. 224; Thomas, p. 83; Merrill, ii. 223; New Orleans in 1820, p. 9; Lewis, N. O. Journ., i. 426.

² Chervin, translation of Wilson, pp. 74–77. He appeals, in this and other works, to Drs. Mery, Dias, Dow, Gillkrest, Amiel, Brown, and others, of Gibraltar, who are cited by Gillkrest and Bancroft; as also to Drs. Lafuente, Melado, Saravia, Balmis, Flores, Gonzales, Genebriera, Lagasca, Arruti, and Velasquez, of Spain; O'Halloran, Yellow Fever of South of Spain, pp. 80–82. Gillkrest, ii. 265, refers to some of those already mentioned, and to Drs. McMullin, Sproule, Welsh, Martindale, Donnett, Humphreys, Hugh Frazer; Med.-Chir. Rev., Jan. 1848, p. 70; Bennett, p. 480, cites, besides the former mentioned, Lee and Shand; Amiel, Edinb. Journ., xxxv. 279; Hugh Frazer, Med.-Chir. Rev., xiii. 338; Arejula, pp. 139, 160–1, 171, 200; Ib., Fever of Malaga, Edinb. Journ., i. 448; R. Jackson (Spain), pp. 89–91, 109, 110; Bally, p. 105; Audouard, pp. 67, 276; Proudfoot, xxvii. 241; Dufour, iv. 50, 57; Rochoux, p. 453; Fellowes, pp. 201, 312, 313; Second Report on Quarantine by Lond. Bd. of H. (1852), pp. 142, 143.

³ J. McGrigor on Fever of Grenada, Sketches, p. 226; H. McLean, pp. 30, 84; Evans, p. 257; Dickson, Report, pp. 143, 144, quoted by Good, ii. 172; Madrid, p. 24; Gillespie,

racter of the disease, in many cases, is upheld by very high authorities. With respect to the fever of Spain, its frequent occurrence there, as Dr. Gillkrest well remarks, entitles the statements of the medical men of that country to great consideration on the point in question. Arejula, who, more than any other, was familiar with the disease—all the epidemics of which he had witnessed—and the results of whose experience are recorded in a volume of great merit, tells us, in the history of the Malaga fever of 1803, that the bark was found useful during the *remissions*. In other places, he is clear on the subject of these: “Sudor ú orina todo bilioso, remision de los sintomas expresados, y de la calentura, con el sudor ó sin él, á las veinte y quatro horas; exâcerbacion al siguiente dia, y remision ó apirexia aparente al tercero, algunas veces al quarto, quinto y septimo, aunque raras” (p. 171). Again he says: “Si las remisiones son regulares, y se observan de veinte y quarto en veinte y quatro horas, como acontece comunmente, conviene menu-dear las tomas de la quina en el tiempo de la remision” (p. 200). Finally, at page 139, he informs us that the disease, without doubt, deserves the name of remittent fever—“*merece sin duda el nombre de calentura remittente.*” Dr. Gillkrest reminds us that the recorded opinions of Dr. Velasquez, of Seville, are fully in corroboration of those of Arejula. Other physicians, contemporaries of the latter, entertained similar views. Dr. Balmis called the disease, as it presented itself during the epidemic of Cadiz in 1800, “a putrid malignant remittent.” Dr. Flores Moreno describes in his work “accessions and remissions.” Dr. Alfonzo de Maria, of Cadiz, says: “When the yellow fever degenerated into intermittent.” In the *Trozos ineditos* of Dr. Salva, of Barcelona, evidence is to be found of the disease having been observed to assume the remittent form.¹

In reference to the yellow fever of the West Indies, a few facts may be mentioned. In speaking of the epidemic of Barbadoes in 1816, Dr. Ralph says: “A most striking disposition to assume a distinct remitting type was observed in this fever. Those who had suffered much from ague, evinced this disposition in the most remarkable manner when the treatment had been regulated by a knowledge of this fact. But, even in the young and unimpaired constitution, when the disease had been seen and treated soon after the period of invasion, it not unfrequently showed distinct remissions. On the other hand, in many of the severe cases, in which remedies were of little avail, the disease appeared to consist of a single paroxysm, the violence of which was such as to occasion extensive structural derangement in one or

p. 43; Gilbert, p. 77; Bally, pp. 95, 226, 254, 482; Huck, in Pringle, p. 198, note; Pinkard, ii. 468; Musgrave, ix. 118–132; Ferguson, viii. 133; Ralph, ii. 69–72; Lempriere, ii. 70; J. Hunter, pp. 65, 68, 73; R. Jackson, Sketch, pp. 51, 54, 60, 71, 75, 93; Pugnet, pp. 354–8, 360–79; Frost, xiii. 30; Furlong, p. 291; Caillot, p. 18; Arnold, p. 33; Bancroft, p. 35; Guyon, Réponse, &c., p. 42; Deveze, p. 55; Thomas Clark, p. 15; Rufz, p. 57; Maher, p. 847, 868–9; Moulin, Diss. sur la Fièvre J., p. 20; W. Humboldt, Rep. of Sanit. Commiss. of New Orleans, p. 133; Mouillé, F. J. Obs. au Cap. F., p. 10; Duprè-petit, Diss. sur la Fièvre J., p. 20; Mabit, p. 15; Lefoulon, p. 64; Bertulus, p. 55.

¹ Second Report on Quarantine (1852), pp. 142, 143.

more important organs, thereby rendering the system incapable of conducting the disease to remission" (p. 69).

The yellow fever which at times prevails on the African coast, has occasionally presented the same character. Of that of Fernando Po, in 1829, Bryson tells us: "The character of the fever was similar to that of Sierra Leone." It was "distinctly remittent in its character, and accompanied by yellow suffusion of the skin and eyes, and black vomit" (p. 70). "The Dryad arrived on the coast on the night of the 7th November, 1830. On the passage out, two men slept on shore one night at Porto Praya, on the island of St. Iago. One was seized with symptoms of fever on the tenth day, the other not until the fifteenth. One assumed a tertian intermittent form, the other ran through its course with all the violence of the yellow remittent of hot countries" (p. 84). The crew of the *Conflict* suffered but little from the disease of the country until July, 1830, when they were attacked, while near Sierra Leone, with fever. Thirty cases occurred, of which eight died on board, and five were sent to the hospital. "The disease appears to have been of the most malignant character. It was remittent, but varied in its symptoms in different cases; in the worst, it was attended with great excitement, and, as it advanced, the skin assumed a yellow colour, interspersed with livid spots. Towards dissolution, in fatal cases, a quantity of dark matter was vomited, while a disagreeable cadaverous smell exhaled from the body some hours before life became extinct." "There can be but little doubt," Dr. Bryson adds, "that all these cases were what is usually termed yellow fever in its worst form" (p. 96). Still, it was remittent in type. The *Buzzard*, in 1839, furnished another example of the kind. The fever was remittent, each case being marked by two distinct daily exacerbations, one in the morning, and the other in the evening—the latter the more severe, and lasting until midnight. The remissions were attended with considerable alleviation of the more urgent symptoms. "Of a crew of seventy-two persons, twenty-seven had the fever in a severe form, and in fifteen it presented all the malignancy of yellow fever, *which it evidently was*." "It was attended with black vomit" (p. 138).

There are not wanting facts, indeed, to show that the disease assumes at times the intermittent character, either doing so from the outset of the attack, and continuing throughout, sometimes unchanged in that respect, or, passing later into that type, from the effect of treatment or the efforts of nature. Such, at least, we gather from the writings of Rush (iii. 73, 214, iv. 13), Deveze (p. 35), Valentin (p. 172), Merrill (ii. 233), Barton (p. 20), Archer (v. 66), Waring (p. 8), and others in this country. Arejula (p. 227) states that the yellow fever of Cadiz terminated not unfrequently with an intermittent, especially at the close of the epidemic. Other writers, as Rochoux (p. 453), Dufour (iv. 50), and Velasquez (p. 13), admit its occasional appearance in that form in Europe, sometimes from the outset, in other cases after some days' continuance. In a memoir on one of the epidemics of Seville, signed by the latter physician, and Drs. Gabriel Rodriguez, Serafine, Adame, and Chicon, and published in the third volume of

Hurtado's Decadas, the fever is said sometimes, though rarely, to present itself, following the type of an intermittent—"Alguna vez, aunque rara, se presenta la calentura siguiendo el tipo de intermitente." In tropical climates, a like statement is made by Pinkard (ii. 90), Gillespie (pp. 43, 130, 146), McGrigor (p. 226), Dickson (*Report in Good*, ii. 173), Musgrave (ix. 118, 126-132), Maher (pp. 849, 868-9), Furlong (p. 291), Dutrouleau (p. 26), Sauty (see *Chervin's Report on Rufz*, p. 70), Spinosa (*Ib.*, p. 757), and Lalle-mant (p. 74). By none, however, has the intermittent type of yellow fever been more pointedly recognized than by Dr. R. Jackson, who, in his *Sketch of Febrile Diseases* (i. 76, 95, 112), admits the periodical form of most, if not all, of the numerous varieties into which he divides the disease. Let the following remarks by a distinguished contemporary writer serve as evidence of the truth of the existence of such cases. Dr. Rufz, in his account of the epidemic of Martinique in 1839, says: "With some children, and with some adult creoles, the disease presented an intermittent type—that is to say, after a severe paroxysm, which lasted from twenty-four to forty-eight hours, there was a notable apyrexia, followed by some paroxysms of a less severe character. These cases were not the most grave; the fever was arrested by sulphate of quinia."¹

Undoubted as the fact may, from the testimony adduced, appear, that the yellow fever assumes, occasionally at least, the remittent—and even, but more rarely, the intermittent—type, not in this city only, but wherever the disease has shown itself; and however prepared we may be, from personal observation, to join in the statement, there can be no doubt that cases of the sort are not as frequently encountered as might be inferred from what precedes. So far from it, experience shows that, in the large majority of instances—especially those of a severe character—the fever takes on at once, and preserves, the continued type; or, if it presents occasional risings and fallings, these modifications are not sufficiently well marked to deserve the appellation of remissions, and still less of intermissions. We have already seen that Dr. Rush himself, who warmly advocated the remittent character of the disease, admitted that it frequently presented the continued type (iv. 13). Other writers regarded that type as the most generally prevalent in this city. Dr. Currie admits that there was some abatement of fever towards morning, but adds, that scarcely in any instance was there so much of it as could properly be called a remission.² Dr. Monges informs us that the yellow fever almost invariably assumes a continued type, exhibiting only obscure and irregular remissions, and that, too, seldom (ii. 56); and the same views are upheld by Dr. Caldwell (*Essays*, p. 104; *Fev. of 1805*, p. 87), Dr. Chapman (ix. 130), and others. In reference to the epidemic of 1820, Dr. Jackson states that when, in the mild form—in which the disease consisted in a mere ephamera—the fever continued from three to five days, it was attended with remissions; but that, in the malignant class, the type was strictly con-

¹ Examiner, iii. 106; Chervin's Report on Rufz, pp. 58, 59, 80-84.

² 1793, p. 20; 1797, Essay on Bilious Fever, Appendix, p. 219.

tinued, while, in the milder cases of the less malignant form, there were only attempts at remissions, or these were imperfect (p. 61).

So far as the many cases that have fallen under my own personal observation are concerned, the *one* paroxysm was certainly continuous. In no instance, when the disease was pure, could any remission, properly speaking, be detected. Nor is it alone with us that the remittent character of yellow fever has been denied, or that the remissions, if admitted at all, have been regarded as rare occurrences, and as being at best always of an obscure and imperfect kind. Dr. Dickson, in speaking of the epidemic of Charleston, in 1838, says: "Then there was the single paroxysm of long duration without subsidence; and when subsiding, returning no more."¹ And in this he is supported by all his predecessors and contemporaries in Charleston,² not a few of whom roundly assert that, for want of attention to this fact, mistakes are often made—persons being pronounced to have had the yellow fever who merely suffered under bilious remittent fever. In other cities of the Union, the same denial of the existence of true remissions has been made, or their existence rendered doubtful from the complete silence observed by writers in regard to them.³

In Spain, including Gibraltar, remissions—though regarded, as we have seen, by Arejula and other writers as an essential attribute of the disease, do not appear to have attracted the notice of different observers. In speaking of the stage of excitement in the very epidemic specially described by Arejula—that of Cadiz, in 1800—Berthe (p. 85) says: "The patients continued in much the same condition during the first two, three, or four days;" and he nowhere makes mention of distinct remissions unless of the one occurring at the stadium (pp. 90–1). If we turn to other descriptions of the disease, we shall find the same silence as regards such remissions, or even a positive denial of their manifestation in a distinct and well-marked form.⁴ Even Dr. Rochoux, in his so often quoted volume, admits the not unfrequent occurrence of remissions or intermissions in the yellow fever of temperate regions; but he does it in a way calculated to prevent us from placing much confidence in the assertion, for he confesses that, whether the disease assume the continued, the remittent, or the intermittent type, the characteristic symptoms, in all cases, are fundamentally the same. He admits that the remittent type, "as it is easy to perceive," is often so confounded with the continued, as not to be distinguished from it; that in regard to the intermittent, it generally be-

¹ Eclectic J., iv. 109.

² Moultrie, p. 4; Lining, ii. 417; Irvine, p. 31; Simons, p. 21.

³ Pascalis, Marseilles J., v. 141; Townsend, p. 176; Tully, p. 302; Revere, iii. 225; Gros, p. 10; Drysdale, i. 41; Girardin, p. 35; N. O. 1839, p. 336; Waring, p. 45; Kelly, xiv. 373; Stone, vi. 554; Dalmas, p. 11.

⁴ Pariset, p. 529; Pym, Edinb. J., xxxv. 41; Ib., Bulam Fever, pp. 60, 228; Vance, in ib., p. 62; Frazer, quoted by Pym; Barry, Med.-Chir. Rev., xii. 539, xiii. 338; Wilson, Med. Lancet, ii. 434; Ib., Translation by Chervin, p. 71; Velasquez, p. 10; Pariset, Obs., p. 29; Proudfoot, xxvii. 241; Burnett, p. 484; Shand and Ross, see Burnett, p. 485; Copland, iii. 141.

gins to manifest itself only three or four days after the disease has exhibited the continued type, and that even then the greater number of the symptoms continue unchanged during the apyrexia—as jaundice, the adynamic state, &c.—while the pulse, during the exacerbation, continues unchanged, in regard to force and frequency, till an advanced stage of the disease.¹ All this is not sufficiently clear to impress us with the conviction of the existence of true remissions and intermissions. Indeed, a clear-headed and well-informed inquirer can hardly help suspecting that all these remarks were intended to confuse the whole subject, and mystify the reader, the more especially as the author closes at last with a description of the disease in only one of the several types the existence of which he had recognized—the continued. To this it may be added, that the same writer who, six years after his visit to Barcelona, admitted, in the way mentioned, the existence of the periodic type in the yellow fever which had prevailed in that city, sounded a different note in a prior publication, written at the very time he was observing the disease. Of the latter, he says: “Although always continued, the concomitant fever exhibits sometimes, though very rarely, irregular exacerbations during the first two or three days; amaril typhus—fever almost always more or less intense at the outset, and always continued.”²

Dr. R. Jackson, who, as already stated, described a periodic form of several of the varieties into which he divided the West Indian fever, found it difficult, if possible, to trace remissions in the concentrated form of the fever of Spain—the one most entitled to be regarded as the true yellow fever. “There is here rarely any recurrence of sensations of cold after the tumults of invasion are past, and consequently the most distinct of the signs which mark the accession of febrile paroxysm is looked for in vain. There is, as in others of the febrile class, a rising and falling of the irritability of the system at given times; but the marks are often obscure—in fact, so obscure that they cannot be appreciated, except by persons who give the minutest attention to the observation of the diseased condition in all its turnings and windings at the bedside of the sick. The fever which prevailed at Xeres, where the character was masked and the symptoms aggravated, proceeded, for the most part, after the tumult of invasion was past, in what may be called a calm and even tenor. There were risings and fallings at certain intervals, but there were no distinct paroxysms and remissions, unless where the disease was mild in itself, or where it had been rendered so by treatment.”³

Not less positive on this subject are many of the highest authorities on the yellow fever of other climes, who either deny strenuously the remittent character of the disease, or regard remissions as of very rare occurrence, or as being obscure, imperfect, and irregular. Chisholm admits the existence of them in what he calls, in contradistinction to his pestilential fever, the remittent yellow fever. But, even in this form of the disease, the periods of remission

¹ *Recherches sur les Différentes Maladies, &c.*, p. 453. 1828.

² *Dissertation sur le Typhus Amaril, ou Maladie de Barcelone*, p. 39. 1822.

³ *Fever of Spain*, pp. 109, 110.

were by no means certain and fixed, or the remissions perfect (i. 247-8). On the other hand, "in the malignant pestilential fever—the true and legitimate yellow fever—remissions were scarcely perceived in any case, and, in general, nothing of the kind happened" (i. 234-5).¹ Moseley, too, remarks, that a remission does not happen in the *causus*, under which name he describes the yellow fever; and, like some of our own writers, he roundly asserts that those who speak of such an occurrence have mistaken bilious remittents for that disease (p. 467), though admitting, at the same time, that "it seldom happens in continued fevers that one, and often two exacerbations are not perceived within the nycthemeron" (p. 468). Dr. Rochoux affirms that the febrile symptoms attendant on the yellow fever of the West Indies assume the continued type, like those of other phlegmasiæ, and that it could hardly be otherwise. "Nevertheless, though never losing its continuity, we perceive, from time to time, some patients experiencing at irregular periods of the day—during the first two or three days—well-marked chilliness (*frissonnements*), renewed three or four times in the twenty-four hours, but during which the skin retains all its heat. The pulse, during the intervals of these sorts of paroxysm, never becomes apyretic. Its frequency has appeared to me, on the contrary, to increase gradually in all these cases, which are usually of a very serious character, and which, when superficially examined, may have led to the belief of a remittent fever" (*Rochoux*, p. 342). Views more or less approximative to these are entertained by Dancer (p. 83), Belcher (p. 251), N. Dickinson (pp. 36, 107), Dwight (*Med. Journal*, xiv. 313), Wilson (p. 10), Savarésy (pp. 267, 292), McArthur (p. 348), Wilson (p. 179), Osgood (p. 28), Stevens (p. 218), Pym (pp. 5, 177), Bally (p. 206), H. McLean (pp. 30, 84), Rufz (p. 12), Lempriere (ii. 61), R. Jackson (pp. 54-5), Desportes (i. 193). Some of these writers deny the prevalence of remissions in all cases. Others regard them as absent in many instances, especially in the more malignant forms of the disease; while others, again, view them as always obscure and imperfect, and scarcely deserving the name of remissions; in fact, as nothing more than a slight abatement in the violence of the excitement. Catel, who has ample experience in the disease, and who has seen it on a large scale, and under circumstances of position which enabled him to watch carefully the various phases in the revolution of the phenomena, from the incipient stage to the close of the attack, by recovery or death, does not hesitate to regard it as a fever of the continued type. To consider, he says, the disease as a remittent, or an intermittent fever, is to commit a serious error, which could have arisen only from a superficial examination of the phenomena. As to him, considering it as a matter of duty to visit his patients frequently, he declares he never saw a case of true yellow fever which called for antiperiodics. Like many others, Catel thinks it very probable that physicians may have treated at the same time intermittent and remittent fevers, and confounded them with yellow fever.²

From all that precedes in relation to the yellow fever of this and other

¹ See also Chisholm's Manual, p. 188.

² Ann. Marit., 1844, iv. 235.

regions, we may infer that in many of the milder cases, particularly when the febrile stage extends beyond the usual time, the disease presents sometimes a slight paroxysmal type, and is subjected to diurnal exacerbations and ameliorations; but that, as a general rule, in well-marked, pure and malignant cases, the type during that stage is continued, and remissions, if they present themselves at all, are obscure, imperfect, and irregular; consisting, in fact, more in diurnal diminutions of the symptoms, or abatements in the morbid feelings, than in remissions, in the true medical acceptation of the term. Such has been the case in all the instances of uncomplicated yellow fever which have fallen under my observation.

Whether the opinion of those who here and elsewhere have maintained that writers who speak of a remittent yellow fever, have invariably confounded together two diseases of a distinct nature, and mistaken bilious remittent for genuine yellow fever, admits of no doubt, is a question I shall not here undertake to examine in detail. Nevertheless, I cannot withhold the remark that when we discover that the advocates of the remittent form of the disease are to be found almost exclusively among those who view yellow fever as identical with, and as only a higher grade of, common bilious remittent fever; and when, moreover, we find that remissions are not often alluded to by those who entertain different views as to the similarity of the two diseases, we may be allowed to regard the opinion in question as entitled to consideration, and to admit that cases that have been pointed out in proof of the existence of a remittent form of yellow fever may, in general, without impropriety, be regarded as belonging more properly to the other variety of fever referred to. The latter, not unfrequently, under peculiar circumstances, both as regards causation or predisposition, presents some points of resemblance to the yellow fever, sometimes to the malignant, more frequently to the mild forms. Cases may even present the black vomit, and other symptoms of like import, and this occurrence may induce physicians to regard them as instances of yellow fever, and, in consequence, to ascribe to the latter a remittent character, while, in fact, they should be viewed in a different light. The black vomit being only an accident, occurring, as we have seen, in many diseases distinct from yellow fever, and even often absent in the latter, cannot be viewed as characteristic of it, or as alone sufficient to demonstrate the true nature of the cases in which it may occur. Besides, it will be seen, on inquiry, that the remission pointed out by several writers, in illustration of the periodic character of the disease, consists in that cessation of febrile excitement which constitutes the metaptosis upon which I have dwelt; while others have evidently in view those momentary sensations of ease and amendment which manifest themselves at times in this and other truly continued febrile complaints, and may properly be regarded as differing from true remissions, unaccompanied, as they often are, by changes in the skin, pulse, &c.

True, however, as all this may be in reference to the larger number of cases, certain it is that instances present themselves, which, though marked by signs characteristic of true yellow fever, and even attaining a high degree of severity and malignity, nevertheless exhibit the remittent type, either at

the outset or at the close of the stage of excitement; and it has been seen that, in mild and ephemeral cases, the same phenomenon is not unfrequently observed to continue throughout the attack. The occurrence in such cases may, perhaps, sometimes be viewed in the light of an anomaly and as the result of the tendency which, under peculiar circumstances, most irritations present of assuming the type in question; an effect more readily to be anticipated in mild cases, when, from the less overpowering or prostrating impression of the poison, the consequent irritation has more ample scope to display that tendency. But, while admitting this to be true in some instances, there can be little reason to doubt that, in a larger number of cases of the fever in which the phenomenon in question displays itself, it must be referred, not to a tendency to remit inherent in the disease, but to a complication of the yellow fever with some one of the several forms of febrile affection of which that tendency constitutes a characteristic element. That the two diseases may prevail at the same time in the same place, and also coexist in the system, are facts too evident to be denied, and upon which I shall dwell more fully in the next chapter.

CHAPTER XXI.

COMPLICATIONS OF THE YELLOW FEVER.

IN the preceding chapters, I have entered fully—perhaps too fully—on the symptomatology of the yellow fever. But, in the description given, the disease has been considered in its purer forms, and no account has been taken of the various modifications which it may experience from the influence of morbid agencies, independent of those to which it owes its existence. The yellow fever is, however, far from pursuing, always, everywhere, and under all circumstances, the even tenor of its course, and without experiencing more or less important modifications from other causes which may operate on the system in conjunction with, or antecedently to, that by which it is produced; while the special morbid agent which gives rise to it, seldom fails to modify, to some extent—when it prevails extensively and with great virulence—other diseases arising from the impress of other causes. Hence arise, on the one hand, numerous complications of the fever with complaints of various kinds; in other words, cases in which, to the symptoms of the fever, are added others indicating the coexistence of some other disease, which owes its origin to the operation of distinct causes; and, on the other hand, those modifications of prevailing complaints occasioned by an impress of the yellow fever cause, which, though not sufficiently powerful to produce the fever to its full extent, is enough so to stamp those complaints with some of its features, and, in the language of Dr. Rush, to make them wear its livery.

Every one is acquainted with the modifying influence of the yellow fever

cause during the prevalence of severe epidemics. It becomes assimilated, in that respect, with other wide pervading diseases, the controlling effects of which have been noticed from the earliest times, even from the days of Thucydides, who, in his celebrated account of the epidemic fever of Athens, so happily imitated by Lucretius (lib. vi.), and Ovid (Metam. 7, cap. xiv.), remarks, that it obliged all other diseases to change their nature by some of its symptoms. "And none of the usual endemic maladies made their attacks during its continuance; or, if they did, soon terminated in this." The same modifying power, as regards malarial fevers generally, and other complaints, has been pointed out in a particular manner by Sydenham, Hodges, Huxham, Sauvage, Stoerk, Lepeeq de la Cloture, Sauvages, Chenot; by our own Rush, and after him by other writers.¹ So far as relates to the fever under immediate consideration, the agency in question has been noticed in times long past, and has not escaped the observation of modern writers. Chisholm found that most diseases degenerated into the yellow fever, and partook very much of this. "Dysenteries suddenly stopped, and were immediately succeeded by the symptoms of the pestilential fever." "In short," he adds further on, "every disease in which the patient was liable to infection, sooner or later assumed the appearance, and acquired the danger of the pestilential fever" (i. 181, 182). Savarésy tells us that, in the West Indies, the yellow fever, exercising its effects in general only on strangers, cannot prevent the occurrence of intercurrent or sporadic diseases, and limits itself to imparting to them a portion of its malignancy and of its physiognomy (p. 293). In the same way we may understand the remarks of Dr. Baneroff—making allowance for some of his peculiar views respecting the true causes of the effects referred to—when he informs us that, in those countries in which the temperature of the atmosphere is usually heated, during certain seasons, to 85° or more of Fahrenheit's thermometer, in the daytime, all febrile affections, however produced—those which sometimes originate from intoxication, and other excesses, from taking cold, or from fatiguing exertions of the body while exposed to the sun, or strong agitations of mind—have a tendency, in consequence of the heat on the human body, to assume the violent and dangerous form, and be accompanied with the symptoms which are usually considered as characteristic of the yellow fever (pp. 6, 7). The yellow fever which prevailed epidemically in Tampico in 1836, exercised a marked influence on all the intercurrent diseases which, in consequence, presented themselves clothed with some of the symptoms of the reigning fever.²

In Europe, during the epidemic of Gibraltar in 1828, Mr. Amiel found that the yellow fever changed the nature of all acute and some of the chronic diseases, drawing them into its vortex, and stamping them, as it were, with its own seal.³ Not a few physicians among us have called attention to the

¹ See Pneumonia, its supposed connection, &c., with autumnal fevers, p. 453; Bally, p. 254.

² Goupilleau, Rept. by Chervin, Bulletin de l'Académie, iii. 308.

³ Edinb. Med. and Surg. Journ., xxxv. 274.

same fact—Vaughan, at Wilmington (p. 20), Drysdale, in Baltimore (i. 371, 372), Davidge (p. 70), &c.

Dr. Rush informs us that, in the epidemics he saw, every intercurrent disease assumed the character and presented some of the phenomena of the reigning complaint (iii. 76, 79). “Such is the case during an epidemic of yellow fever,” says Dr. McFarland, of New Orleans; “let a man be attacked with almost any febrile disease when yellow fever prevails, and it is certain to be ushered in or accompanied with some of the general outlines of yellow fever; while itself, indeed, on its first invasion, resembles nothing more than a violent cold, and hence its insidious fatality, so often produced by delay.”¹ Dr. Barton, too, in his report on the sanitary condition of New Orleans in 1853, touches on this subject. After remarking that an epidemic disease is known to prevail when a large number of cases of disease of the same type and character break out, either simultaneously or within a brief period, over a considerable extent of a city or country, wearing one general livery, and evincing and maintaining a sway over all prevailing diseases, he adds that “the statement of this proposition will carry conviction of its truth to all those who witnessed the characteristics of the disease during that season, where forms of morbid action, that were not suspected to be yellow fever, from wanting its prominent symptoms, were suddenly terminated by black vomit.”²

Cases of this kind belong, perhaps, more specially to the history of the diseases which are thus modified by the yellow fever cause than to that of the yellow fever itself. But it can matter little whether the effects under consideration be the result of an engrafting of that disease on complaints already existing, or of the supervention of one of these in a system affected with the poison of the other, or of the simultaneous operation of the causes of both. In either event we have to deal with cases of a hybrid complaint—not with peculiar forms of either of the morbid states entering into the compound. The only difference is that, in some instances, we have the yellow fever engrafting itself on some other disease, playing the part of a parasitic complaint, and imparting to the latter a greater or less number of its phenomena; in others, the symptoms of the fever will stand in bold relief, and be associated with some which form no part of its natural composition, but modify somewhat its course—mask, to a certain extent, its true character, or perchance change its type; while in a different set, again, the two diseases may keep their ground, for some time at least—for it seldom happens that the yellow fever does not at last gain the mastery over its antagonist, not only in this category of cases, but in all—modifying each other in various ways; sometimes ending together by the death of the patient, or surviving each other. But, I repeat, whatever be the manner in which the assemblage of the phenomena observed is brought about, the result

¹ List of Interments in all the Cemeteries of N. O. from the 1st of May to the 1st of November, 1853, p. 10.

² Report of the Sanitary Commission of N. O. on the Epidemic Yellow Fever of 1853, p. 262.

is a complication of diseases, in which the yellow fever, though deprived of its purity, plays an important part.

It can scarcely be necessary to remark that the number and variety of such complications, by which the purity of the disease is necessarily much impaired, and which, from inattention on the part of careless observers, and a disposition to jump at conclusions from insufficient data, and after superficial examination, too often exhibited by so-called pathologists and etiologists, have often been the source of much confusion in the history of the fever, are considerable. For there is scarcely a disease, whether zymotic or phlegmasial, which may not, on some occasions, associate itself with the complaint in question; sometimes one, sometimes another, leading the way. Dr. James Clark states that in 1793, at Dominica, children, adults, and old people, labouring under smallpox, were attacked with the yellow fever about the time the secondary fever generally came on, whether the disease assumed the confluent or simple form (p. 19). In Philadelphia, the fever of 1798 was preceded by a considerable spread of smallpox. "When, as we learn from Dr. Caldwell, the former disease appeared, and assumed an epidemic form, the latter became more malignant and fatal, and was marked, in many cases, by genuine black vomit. It yielded, at length, completely to the predominancy of the epidemic, and entirely disappeared long before the termination of that complaint."¹ Dr. Rush informs us that, in 1799, the fever—to use his own words—blended itself with smallpox. Its union with this disease "was evident in two patients in the City Hospital; in each of whom the stools were such as were discharged in the most malignant state of the fever" (iv. 57). Dr. Drysdale, in his account of the epidemic of Baltimore in 1794, states that the smallpox, which was prevailing before it broke out, did not vanish, but retained its ground. "The two were accompanied with symptoms common to them both, and had the eruption attended the yellow fever, instead of a yellow skin, or *vice versâ*, they might have generally been classed under the same name."²

Dr. Rush, from whom we learn that the scarlatina prevailed extensively in this city in June and July, 1802, mentions a case in which that disease was blended with the yellow fever. The scarlatina had imparted to the fever a general redness of the skin, which induced the patient, a female, to believe it was that disease, and to neglect sending for medical relief for several days. She died with red eyes, a black tongue, and the usual symptoms of yellow fever (iv. 78). The same thing had taken place in 1794, when the fever "blended itself with the scarlatina" (iii. 214). The measles also partook more or less of the character of the reigning epidemic. (*Ibid.*)

The yellow fever is not unfrequently complicated with symptoms indicative of the existence of visceral and other phlegmasiæ. Pneumonia, pleurisy, and peritonitis are sometimes detected during life, and traces of them found after death; or the fever imparts some of its features to those inflammations in the way we notice in regard to ordinary autumnal fevers. This complication oc-

¹ Med. and Phys. Mem. (1820), pp. 168–9.

² Coxe's Med. Mus., i. 372.

curs even after the cessation of the epidemic, probably in persons who had imbibed the seeds of the disease. Dr. Rush, in his account of the epidemic of 1793, notices it, remarking that the complaints that followed had this peculiarity, "that many of them had several of the symptoms of the yellow fever, particularly a puking of dark matter (which he regarded as bile), dark-coloured stools, and yellow eyes. "Mr. Samuel D. Alexander, a student of medicine from South Carolina, who was seized with pneumonia about Christmas, had with it a yellow eye, a dilated pupil, and a hard pulse, which beat only fifty strokes in a minute. His blood was such as I had frequently observed in the yellow fever." He relates several other cases of the same kind in which the blending was very apparent (iii. 102). Dr. Rush was too acute an observer to be surprised at these anomalies, for he had been taught by Sydenham that the epidemics of autumn often insinuate some of their symptoms into the winter diseases which follow them.

In some instances, rheumatic symptoms open the scene, and mask the nature of the disease. Dr. Rush mentions the complication in his account of the epidemic of 1799 (iv. 57). An instance of the kind fell under the author's observation during the past season. The patient came from Savannah, where he doubtless imbibed the seeds of the fever. During a few days, he presented no other symptoms than those of acute articular rheumatism. At last the phenomena of yellow fever made their appearance. The patient lost soon all power over the muscles, and died with black vomit and its usual attendants. Dr. Blair, among others, alludes to the occurrence of these complications during the epidemic of Demerara—mentioning that peritonitis and pneumonia failed to exclude the invasion, and that persons under treatment for rheumatism were attacked, often fatally.

Dysentery is another not very unusual source of complication. To say nothing of the circumstance that colitis was found by Louis in some of his dissections at Gibraltar in 1828 (p. 114), as the morbid changes are not stated in connection with the symptoms which presented themselves during life, we find the complication described by a good observer, M. Maher (p. 887). Both Dr. Rush and Dr. Drysdale allude to the combination in their accounts of the epidemics of Philadelphia and Baltimore in 1794.¹ It has often been noticed in New Orleans, and is especially referred to by Dr. Fenner in his history of the fever of that place in 1846.)² Dysentery constituted a prominent feature in the case of a Mr. Crowell, who died here of the disease in the summer of 1853. He was attacked on the 16th of August, and died with decided black vomit on the 19th.³ The same dysenteric phenomena were exhibited in the very first case of that season—constant desire to go to stool, attended with tormina, tenesmus, and discharges of sero-mucous matter tinged with blood. The case ended fatally with black vomit in five days (*ib.*, 248–9). In reference to this case, my distinguished friend, Dr. W. T. Wragg, of Charleston, South Carolina, to whom its history had been referred by me prior to publi-

¹ Rush, iii. 213; Drysdale, Med. Mus., i. 370.

² N. O. J., iii. 464.

³ Trans. of College of Phys., N. S., ii. 249.

cation, writes me, under date of November 2, 1854: "My object in troubling you with this communication is to inform you that I have had several such cases under treatment this summer. In one case occurring in an Irish woman, who died with indisputable symptoms of yellow fever, ulcerations of the dysenteric kind were found in the intestines. In others, the graver disease did its work too fast to allow time for such traces to be left. But, in all, there were the clearest and most unmistakable proofs that the fever had been ushered in by the dysenteric symptoms."

In a report on the cases treated in the Roper Hospital by Dr. Wragg, and published since the above was written, he refers to the complication in a passage which, at the risk of repetition, I must transcribe, with a view particularly to show the importance attached to the facts in question. "Several cases, also, were complicated with dysentery. There were six of these, and, as was stated above, one died. In the stomach of this patient, black vomit was found on *post-mortem* examination. This fact is extremely interesting, proving, as it does, most satisfactorily, that genuine yellow fever is met with in combination with dysentery. For, upon the possibility of such a blending of these two sets of symptoms, rests the decision of the question as to whether the epidemic of yellow fever in Philadelphia in 1853 was imported or indigenous, a case of this kind having occurred in that city previous to the arrival of the famous barque Mandarin."¹

During the same season at Charleston, other cases were noticed in which the attack commenced with diarrhœa, and after the first few hours had passed, the symptoms of the yellow fever appeared and kept on their uninterrupted course till death closed the scene. The cholera influence was probably no stranger to these effects, inasmuch as the tendency to intestinal discharges was evinced in a manner very unusual in the disease. Active purgatives often produced an obstinate serous diarrhœa, accompanied, in many instances, with the most distressing tormina, which could only be relieved by opium and astringents. Indeed, the effect resulted from a moderate perseverance in the use of purgatives. The evacuations were purely serous, and had no other tendency than to weaken the patient and hasten the fatal result.

The diarrhoetic complication is not one of unfrequent occurrence, or at least has been seen sufficiently often to justify its not being regarded as one of an extraordinary character. The readers of this volume need scarcely be informed, that the yellow fever poison has been found to coexist with that of Asiatic cholera. This was particularly the case in the city of New Orleans in 1832, where the two diseases prevailed simultaneously to a considerable extent, complicating and modifying each other in the most marked manner.² When epidemic cholera appeared in New Orleans, says Dr. Drake (ii. 303), "yellow fever was prevailing, and soon put on so

¹ Charleston J., x. 83.

² Halphen, Mémoire sur la Cholera Morbus compliqué d'une Epidémie de Fièvre jaune qui a régné simultanément à la N. O. en 1832. Paris, 1833.

many symptoms of that disease, that many cases were only recognized as such by the fatal hemorrhage and black vomit of the closing stage." It may not be uninteresting to remark, in reference to this subject, that of the fifty-seven observations detailed by Dr. Halphen, the chronicler of this event, eight or ten portray the combination of symptoms produced by the complication, and the modification in each of the two diseases resulting from the co-operation of the two poisons. In all, we see that the remedial measures employed in the treatment of the yellow fever favoured the development and enhanced the danger of the other; that the supervention of the cholera in cases of yellow fever tended to moderate the force of the disease; and that, in proportion as the former progressed, the latter receded. In other words, the yellow fever yielded to the influence of the cholera. In no case was a contrary result obtained, a fact from which Dr. Halphen concludes that the cholera poison is more deadly than that of the yellow fever. Take the following case as an example of the complication. The patient, two weeks in the city, aged 17 years, and of a sanguine temperament, was attacked with symptoms of yellow fever on the 29th of October. At 10 A. M., on the 30th, he presented the following symptoms: Pulse full and 120; face red and animated; tongue red on edges and pointed; acute pain at the epigastrium, and in the head and loins; eyes red and injected; urine red and scanty; considerable thirst; constipation. At 6 P. M., pulse 94; urine less red and more abundant; pain in the head somewhat less violent; that of epigastrium has disappeared; rachialgia the same; tongue less red, and thirst more abundant. On the 31st (6 A. M.), pulse 120; yellow coloration of the face, chest, eyes, and *alæ nasi*; urine more abundant, and of natural colour; tongue moist, and less red. The patient has had two serous evacuations after the administration of an injection. At 6 P. M., pulse natural; patient feels better; has had another serous evacuation per anum; tongue slightly coated; neither thirst nor pain; jaundice deeper than in the morning. On the 1st of November, Mr. Halphen found the patient *in articulo mortis*. He had had twenty-two evacuations, and some choleric vomiting since the last visit, suppression of urine, &c. He died in a few moments after (p. 123).

The true yellow fever not unfrequently associates itself with that form of fever denominated *causus*, *synocha*, or inflammatory, and which prevails in all hot latitudes, and in the hot seasons of temperate or even of northern regions, and is brought on by intemperance, great fatigue after being overheated by the sun's rays, sudden diminution of temperature, violent agitation of mind, &c., but principally by insolation—the whole irrespective of the real causes of yellow fever. In such complications, we have the phenomena of high arterial reaction characteristic of this latter disease—hot skin, full bounding pulse, sily and cupped blood, and signs of inflammatory irritation of the brain and other internal organs; combined, in the last stage, with jaundice, black vomit, and other symptoms marking the closing scene of the yellow fever. That such cases are often confounded with some one of the ordinary grades of the latter disease, there can be no doubt; and by more than one writer instances of the kind have been adduced in exemplification simply of the existence of a special

form of yellow fever. In saying this, however, I would not wish to be understood as denying the existence of an inflammatory variety of yellow fever; but I have no hesitation in stating that in localities where the *causus* prevails, and among individuals who, from constitution and habits, are prone to its attack, cases such as I have referred to are the result of the co-operation of the causes mentioned and of the poison of true yellow fever, and not a mere modification of the latter.

Besides the diseases mentioned, the yellow fever has been observed to engraft itself on dropsy, coryza from hydriodate of potash, ptyalism, delirium tremens, lead colic, anæmia, sarcocele, scalds, bruises and flesh wounds, fractures, erysipelas, ophthalmia, syphilis, strictures, eezema, mosquito wounds, hooping-cough, and phthisis (*Blair*, p. 70). Furthermore, the yellow fever becomes frequently engrafted on various forms of fever, or these are often engrafted upon the yellow fever. In 1777, Closset observed it associated with a putrid typhus.¹ So, also, did Drogart.² The yellow fever which prevailed epidemically in Charleston in 1852, was accompanied by, and blended with, the dengue or breakbone fever. But, more commonly, especially, of course, in malarial regions, the complication takes place with autumnal fever, and results generally in the combination of the phenomena of the former, modified by a change of type—the cases ending in death with jaundice and black vomit, or closing in recovery, followed sometimes by paroxysms of periodic fever. The amalgamation is principally found to occur during mild epidemics of yellow fever, when the disease cannot, from want of energy in the poison, take such a firm hold of the system as to chase away all other morbid impressions, and reign supreme. Or it may occur in the very early period of an epidemic, or just before its outbreak, when the poison, though formed, has not yet acquired its full sum of energy; or at the decline, when it has lost a portion of that energy. Or, again, it may occur on the extreme boundaries of an infected district, where the malaria of common autumnal fevers is fully developed—as marked by an extensive prevalence of such fevers—and where the poison of the yellow fever is too diluted to produce its genuine and legitimate effects. In all such instances, the two diseases often struggle for the mastery; they mix together in every possible way and degree, and while in many, paludal fevers are completely superseded by the other, or change their type to the continued, and only reappear in their regular garb after the cure of the yellow fever, in others, the two diseases coexist in a more or less marked manner. The suffused face and bloodshot eyes; the frontal and orbital pain; the distressing rachialgia; the crimson-edged tongue; the bronzed icteric skin and the black vomit—are seen engrafted, as it were, on the fevers of the season, while the case assumes the periodic characteristics of these. In some, the paludal precedes the yellow fever; in others, the periodic element, the product of previous paludal intoxication, shows itself only after the attack has commenced, or they originate together. By proper treatment, the periodic symptoms may sometimes be removed, while those of yellow fever

¹ Referred to from *Brit. and For. Med.-Chir. Rev.*, i. 72.

² *Diss. sur le Typhus*, p. 14.

pursue their course. In fatal cases, death is usually preceded by the phenomena attendant on the closing scene of the yellow fever. In some instances, the yellow fever, when cured, may be followed by a succession of periodic paroxysms.

In the chapter on the type of the disease under consideration, it was stated that the occurrence of cases described under the name of yellow fever, but presenting the periodic character, had long been noticed, both in temperate and tropical regions. It was stated, at the same time, that though there was reason to believe that, on many occasions, the true nature of the disease had been misunderstood, and that the physicians who described them as cases of periodic yellow fever had, in reality, to deal with an aggravated form of bilious remittent fever, and not with the genuine disease—such physicians being mostly advocates of the pathological and etiological unity of both—yet, in other instances, it was not possible to deny the yellow fever nature of the cases referred to, and to doubt the fact that the disease could at times, and under particular circumstances, assume a periodic character. It was seen, however, that most of the authorities mentioned in connection with the subject—whether those who might be supposed to have misunderstood the nature of the cases alluded to by them, or those who had evidently in view instances of true yellow fever—regarded such manifestations of periodicity as an evidence of the fact that the intermittent and remittent types are as much attributes of the yellow fever as the continued; that the disease may sometimes assume one of those types, and in other instances present a different one, without, on that account, losing any portion of its purity, or changing its character. In other words, according to this theory, the yellow fever may present itself under three forms, each marked by a separate type—the continued, the remittent, and the intermittent. But, whatever be the type, the disease is always one and the same. It is produced by the same cause, varying in point of virulence, but acting without admixture of any other morbid agent; and, whatever be the assemblage of phenomena produced, we must see in that assemblage nothing but the *one* disease—pure and unsophisticated yellow fever. We must ignore all idea of complications in cases marked by a compound of symptoms appertaining to that disease and common malarial fevers, and regard the occurrence of such a compound as a proof that the two fevers are identical in both a pathological and etiological sense.

But a different view may be, and has been, adopted in relation to this subject; and in the chapter on type, to which I have just referred, the combination above mentioned was attributed not to a tendency to remit or intermit inherent in the yellow fever, but to a complication of that fever with some one of the several forms of which periodicity constitutes a fundamental and characteristic element. The idea of this coexistence of yellow with common autumnal fevers is certainly not new. It was entertained and ably enforced, more than a century ago, by Pouppe Desportes, who devotes a separate chapter of his work on the diseases of St. Domingo to the consideration of the subject, and especially to the simultaneous existence of the former, or *mal de Siam*, as he always denominates it, with the double tertian, which he regarded as a form of fever peculiar to the colonies, and constituting alone three-fourths of

the febrile complaints of the above-mentioned island.¹ Therein, and in a former part of his volume, he dwells on the impress made by the former on all prevailing complaints, and on the fact that by engrafting itself upon them, it forces them to simulate it. Desportes relates several cases, which leave no doubt as to the correctness of the opinion that they were the result of the complication in question.

Other writers on the fever of the West Indies have adopted similar views on the subject, and dwell, with greater or less emphasis, on the co-operation of the two poisons, especially the engrafting of that of yellow fever, on the one giving rise to common paludal diseases.² Rochoux, in particular, enlarges on the necessity of paying regard to these complications, which, he thinks, present themselves during all epidemics occurring in paludal localities, and rates roundly the successors of Pouppé Desportes for having passed in silence the remarks of that judicious observer on the subject (p. 216). Like the latter, Rochoux relates several cases to prove the existence of the complication. The following description, borrowed from Lempriere, will give a tolerable idea of it, as it is usually observed in the West Indies: "In this, the first attack is marked by the usual symptoms which usher in the remittents, except that the affection of the head is more severe, and the eyes wear a suffused appearance; remissions and exacerbations proceed alternately, as in common remittent, and bark, in large doses, generally is retained during the remissions, from which the inexperienced are wont to draw favourable conclusions. But, about the third or fourth day, and sometimes later, such symptoms occur as denote the greatest danger; the eyes become muddy and suffused, the countenance despondent, and the neck, and afterwards the whole body, dyed with a yellow suffusion; great irritability of the stomach, and oppression about the præcordia; delirium, or more frequently coma, and many other symptoms of the genuine continued fever supervene; and sometimes the true black vomit marks this stage of the disease, and the patient is carried off, at the end of a convulsion, on the fifth, more frequently on the seventh, but sometimes at so late a period as the ninth day. These symptoms, however, are in general treated more successfully in this variety of the disease than in genuine cases of continued fever, and patients sometimes recover from the customary mode of treating the remittents; whereas, in the true continued fever, the stomach is usually so irritable as not to retain such remedies as are employed in cases of the remittent, were they proper for the removal of the disease."

The intermittent malarial fever, according to Blair, is so powerful at Demerara, that the yellow fever of 1837-'42 could not entirely supersede it. Hence, it was sometimes found "engrafting itself on the weakened convalescents from yellow fever, and in a few cases it seemed to dispute possession of the victim; and, in some, modified the procession of symptoms" (p. 22). During the epi-

¹ *Maladies de St. Domingue*, i. 230.

² Lempriere, ii. 70; Osgood, pp. 27, 28; Imray, *Edinb. Journ.*, lxiv. 337; Rochoux, *Rech. sur la F. J.*, p. 192; Blair, p. 22.

demic of yellow fever at Tampico in 1836, already referred to, intermittents, in particular, assumed this complicated form; for, during the continuance of the epidemic, they almost invariably presented, in addition to their legitimate symptoms, one or more of those appertaining to the yellow fever.¹ From Dr. Pennell, of Rio Janeiro, we learn that for some two or three years previous to 1849-'50, when the yellow fever broke out with great violence in that city, it was generally observed there "that fevers presented a different type to what they had hitherto done; and occasionally a case was seen attended by all the pathognomonic symptoms of that disease, and was declared, by the physicians in attendance, to be such."²

Dr. Lallemand, in his account of the same epidemic, makes much the same remarks relative to the monopolizing influence of the fever. "Whatever was the febrile disturbance, it with great facility took the character of yellow fever. This peculiarity of diseases to take a character so special—this necessity of their getting a form so widely extended—was the consequence of the state of the atmosphere, of the epidemic genius, &c." "It is for this reason that other diseases under the unlimited dominion of the epidemic appeared not to exist." "E' por isto mesmo que outras doenças, debaixo do dominio illimitado da epidemia, parecião não existir."³ Dr. Lallemand describes a remittent, and even an intermittent form of yellow fever, the admitted results of the complication referred to.⁴

In Europe, the occurrence of this coexistence has not been recognized, or referred to as such. Rochoux, the strenuous supporter of it in regard to the yellow fever of the West Indies, denies the possibility of it so far as concerns the fever of temperate regions, which he describes under the name of amaril or yellow typhus, and considers as specifically different from the other. But the views of that distinguished but biased writer, respecting the disease in question, are based exclusively on the result of his observations at Barcelona in 1821; and, as the epidemic of that year was one of great severity, and well able, on that account, to monopolize the fullest sway over the ill-fated city, he might well be understood to have met with no such instances, while his theoretical predilections prevented him from discovering them elsewhere. Let any one, however, examine the facts cited by Spanish and other authorities in proof of the remittent and intermittent types of the yellow fever of their country—some of which have been referred to in the preceding chapter—and he will, I think, have no difficulty in discovering in them evidences of the existence of the complication in question. Turn we now to this country, and we shall find that instances of the kind are frequently encountered, and the mode of their production well understood. Dr. Wood, of this city, after stating that the febrile stage of the yellow fever is continuous, like that of smallpox or measles, and contrasts, in this respect, with that of bilious fever, which has a tendency to remit or intermit every day, or every other

¹ Goupilleau, *op. cit.*, p. 308.

² Rep. of Sanit. Commiss. of New Orleans (1853), Test. of Dr. Pennell, p. 150.

³ Observações acerca da Epidemia de Febre Amarella, &c., p. 43.

⁴ *Op. cit.*, pp. 72-74.

day, to the end of the disease, adds: "It is very possible that the causes of the yellow and bilious fevers may sometimes co-operate, and thus produce modified cases. A patient may be attacked with yellow fever while labouring under a remittent or intermittent, which will thus assume the characters of the former; or a predisposition to intermittent may exist, which will exhibit itself after the cessation of the more violent disease" (i. 304, 1st ed.).

Dr. Kelly, of Mobile, remarks that the supervention of yellow fever on an intermittent or remittent, with the supposed mingling, in this manner, of their types, has been adduced as an evidence of the identity of the two diseases—the only difference being in their intensity. Such an argument, he says, is about as valid as it would be to infer the identity of smallpox and measles from the fact that the first may supervene upon the latter.¹ Dr. Nott, of Mobile, whose authority on these matters cannot be questioned, tells us: "When yellow fever prevails, as I have seen it, in a milder form than what we term epidemic, it is invariably seen more or less blended with the intermittents and remittents of the environs; they are mingled in every possible grade." "In 1844, many of these cases occurred in Mobile. In this year, there were only forty deaths from yellow fever, and no epidemic constitution of the atmosphere was established; the two diseases, struggling for mastery with nearly equal force, were blended in every conceivable degree in different subjects—sometimes the periodic, at others, the true yellow fever type having the ascendancy; the former preponderating, particularly in the suburbs, near the marshes. The cases were sprinkled over the whole town, without being confined to any particular focus."² Another good authority, Dr. Lewis, in an account of the yellow fever, as it prevailed in the city of Mobile from 1819 to 1843, inclusive, states that, in the latter year, simple remittent fever prevailed extensively in the southern portion of that city, mostly among the natives and acclimated. He there attended fifteen cases of remittents and intermittents assuming the rank and grade of yellow fever. None of the individuals thus affected were acclimated. The number of these cases, during the course of the season, is estimated at one hundred, of whom fifty died. From the account given of them, we can have no doubt that, aside from the periodic element which was the result of the paludal locality where they occurred, these cases differed in nothing from those of true yellow fever. While such was the character of the disease in that district, no instance of grave fever of one paroxysm was then seen. At Spring Hill, seven miles from the city, three cases of the kind occurred in young men who had been on several visits to the city. The prevailing disease of the Hill was intermittent. Dr. Lewis did not consider them, as others did, as simple intermittents, running, under atmospheric influence, into black vomit, but as primarily yellow fever modified by the paludal poison, or of a mixed kind.³

In a subsequent publication, Dr. Lewis enters into some details in relation to this subject, which may very appropriately find a place here.

¹ Am. Journ. Med. Sci., N. S., xiv. 376.

² N. O. Journ., iv. 584, 585.

³ Ib., i. 292-3, 413.

Every summer and autumn intermittent and remittent fevers prevail in the suburbs of Mobile, whilst those living in the middle part of the city seldom have this character of disease, unless in sickly years, and then the cases are confined to those who are acclimated, while strangers unacclimated suffer with the more violent form of yellow fever.

In the year 1843, very few persons residing on the outskirts of the city escaped periodical fevers, quotidian being the most usual form. That portion of the population who were acclimated had but one or two paroxysms, whilst those who were not had chill and fever, complicated with yellow fever. When medical men were treating what they supposed to be simple intermittent, and were not apprised of the real character of the disease until they were startled by the appearance of black vomit, as was frequently the case, they consoled themselves by stating that a simple case of chill and fever had, *under atmospheric influence, run into black vomit.*

“Now, what are the facts and conclusions to be drawn therefrom? Although the peculiar characteristics of chill and fever were very prominent, so much as to conceal from the observation of the incautious physician the real nature of the disease, yet there were signs by which the hidden demon could be traced out. During the apyrexia, there were the peculiar pulse, some of the restlessness, and that peculiar appearance of the eye, or a glimmering of that unmistakable physiognomy peculiar to the yellow fever, which, once seen, can never be forgotten, all of which prominent traits merit the highest consideration.

“Independent of these signs and symptoms, there are other facts which force on us the conviction that these cases were complicated, from the initiatory chill, and we ask for them a careful consideration.

“We allude to the duration of illness, and the period at which black vomit made its appearance. Under the appropriate head it will be seen that grave malignant yellow fever, well marked from the onset, terminated between the third and seventh day. Of 28 fatal cases of intermittent and remittent yellow fever, all died within the eighth day after the initial chill; two had black vomit in the third paroxysm (quotidian), and died on the fourth morning. Those that recovered passed through the critical, or collapse stage of the yellow fever, on the fifth or sixth day. Inasmuch, then, as the result of the distinct morbid action, constituting yellow fever, proves that five or six days are requisite to the changes which must precede the advent of this critical symptom, we cannot avoid forming a similar conclusion, that the same morbid process was going on in those intermittent cases which had a corresponding termination.

“Every pathological fact yet fully demonstrated, and the accumulated experience of time, is in support of the position that peculiar changes in the system occur previous to black vomit.

“In 1844, remittent yellow fever prevailed to some extent in this city; the cases well marked and of long duration, ranging from five to twelve days; they were attended with hemorrhage from the gums and nose in a large pro-

portion of the cases, presenting a low condition of the body, but most generally they recovered.”¹

For several years prior to the great epidemic of 1853 in New Orleans, it was observed that the disease, though prevailing at times extensively, and causing considerable mortality, did not manifest the monopolizing and absorbing tendency it had done in former days. Hence, we find Dr. E. H. Barton, of that city, remarking that from the experience of what had happened, he was led to unite in sentiment with those professional men who thought that the yellow fever was gradually losing its individuality, and becoming blended with ordinary fevers. “Once very distinctly marked—now, many cases puzzle the most experienced in its diagnosis.”² In a more recent publication, Dr. Barton expresses himself in more positive terms, remarking that, for some years back, the disease has been blending itself with the ordinary diseases of the country. This opinion, he adds, he formed after due deliberation.³ Of the frequency of this complication in New Orleans there can be no doubt. Though accounted for on very different principles by some of the physicians of that city, and especially by Dr. Fenner, who has recently occupied much time in the investigation of the yellow fever, and published several essays and reports on the subject, the facts recorded and the statements made by them, establish the reality of the occurrence. Speaking of the epidemic of 1849, Dr. Fenner says that “cases which they (some physicians) had no idea of pronouncing *yellow fever* in the commencement, proved to be so in the sequel” (i. 45). In another place, we are told that, in the healthiest years, the intermittent type predominates over all others; but, during the sickly years, in the country, it runs into remittent, bilious, and congestive—whilst, in the city, it runs into yellow fever (i. 109). “Rarely does a summer pass in which we do not hear of some intelligent and experienced practitioner being perfectly astonished at seeing what he has pronounced a case of intermittent or remittent bilious fever, terminate in black vomit or other hemorrhage” (i. 111). Plain cases of yellow fever, if not promptly cured, often taper off into intermittents (*N. O. J.*, iv. 538). The same statement is made in other places (p. 117, &c.). In his account of the epidemic of 1850, which was mild, and did not get the mastery over other fevers, Dr. Fenner says: “In the epidemic now under consideration, the slight initiatory chill, and the great heat of the surface that soon followed, the flushing of the face, the pain in the head, back, and limbs, the remission that generally took place within the first twenty hours—and, above all, the frequent occurrence of *bilious vomiting*, combined to make almost a perfect representation of what is recognized as bilious fever all over the southern country; whilst the extraordinary prostration that followed even mild attacks, the frequent occurrence of slight hemorrhages, and the occasional termination with black vomit and yellowness of the skin, afforded indisputable evidence of yellow fever” (ii. 87). In all these cases, Dr. Fenner

¹ New Orleans Med. and Surg. Journ., September, 1847, p. 154.

² Report to the Bd. of Health, Fenner's Southern Rep., i. 84.

³ Report to the Louisiana State Med. Soc., p. 30.

sees nothing like a complication—one fever does not engraft itself on another, take its place, or combine with it. He believes it all the same disease, differing only in grade and stage, yellow fever being only one of the forms or types of endemic malarious fevers (i. 111). Every one, of course, is entitled to the liberty of giving to occurrences he witnesses any explanation he may think fit. In this matter I am forced to differ from Dr. Fenner, on grounds which will be pointed out in a subsequent chapter.

Professor Dickson, of Charleston, long ago described this actual intermingling of the types of fever in malarious climates, and their supervention one upon another. "In any of the localities where yellow fever is endemic, a remittent or intermittent may, at any stage of its progress, assume the malignant character of the prevailing pestilence, when locally epidemic. In the summer of 1817, many northern and foreign sailors had been induced to go as boatmen up our rivers. Considerable numbers of them were brought into our hospitals with 'country fevers,' both remittent and intermittent, which, as soon as yellow fever became prevalent, ran into that epidemic; the fever becoming continued, and the black vomit ensuing."¹

In the letter, already cited, with which I was honoured by Dr. Wragg, of Charleston, the writer says: "I may also state, in corroboration of the position upon which, I think, we agreed in our conversation—*i. e.* that complications could not be said to destroy the character of the disease—that we had in the hospital several instances of complication with the intermittent fever. Patients who had been subjects of the latter, upon being attacked with fever and coming into the hospital, would, for a certain time, present the intermittent character in their disease; but this would finally give place to the more dangerous fever, and the less be merged in the greater. In some of these cases, when we succeeded, by free use of quinine, in arresting the intermittent fever before the time for the yellow fever symptoms to disappear, these latter would resume their interrupted march, and these usually ended favourably. In others, the intermittent form continued till the proper febrile stage of the yellow fever had passed, and then, upon the cessation of the last intermittent paroxysm, the bronzed, jaundice, and comatose state peculiar to that period of the yellow fever appeared in the place of the other, and the patient died." In the report already cited (p. 83), Dr. Wragg, in reference to these cases, says that they "wore the blended livery of the two diseases, and it was not possible to confound them with those in which yellow fever ran its course, continuous, from first to last, and absolutely uncomplicated with the intermittent element. They all resulted favourably under the vigorous use of quinine except one. This case had been exposed to the influences which are known to produce both forms of the fever in the most dangerous degree. The patient had contracted his intermittent fever in Georgetown, and his yellow fever in the most infected part of the city."

Dr. Dickson calls attention to a circumstance in relation to the epidemic

¹ On the Blending and Conversion of Types in Fev., Trans. of Am. Med. Assoc., v. 152-3.

of Augusta (Georgia) in 1839, which has been observed in very many places besides. The fever which broke out about the third week of August, though of a more fatal character than that usually prevailing there at that season, and necessarily presenting some symptoms not generally encountered, was nevertheless regarded by the physicians of the place as varying "in no material features from those frequently exhibited in the ordinary summer and autumnal fevers of all southern climates." A few days later, the Board of Health report that "the disease is the bilious remittent of the season and climate *aggravated* by some local cause." A physician of eminence and experience writes that the disease is not yellow, but bilious remittent fever; but that, "in a small portion of the city, a considerable number of cases were so modified as to render them highly dangerous;" and were "properly denominated congestive cases of bilious fever." Another physician, conversant with all forms of southern fevers, writes that so far as his observation extended, "the cases had the aspect of bilious remittent fever, assuming, in the latter stages, the typhoid type;" yet, as Dr. Dickson adds, before the termination of the season, there was no dissentient voice to the universal admission that the epidemic was, and had been from the commencement, yellow fever. "But how complete and thorough must have been the *blending* of features in this pestilential invasion, thus to disguise, under the mask of common and familiar symptoms, a new and terrible enemy." (*Op. cit.*, pp. 151-2.)

In Wilmington (Delaware), in 1802, according to the testimony of Dr. Vaughan, after the middle of September, the subordinate forms and grades of fever, when not arrested within forty-eight or seventy-two hours, invariably passed on to the malignant grade of disease. "No matter how slight the attack, nor who the subject, the livery of pestilence sooner or later appeared."¹ Much the same results were obtained in Baltimore in 1794. In many instances, the yellow fever commenced under the form of a periodic fever. The case presented the phenomena of the latter till the close of the second paroxysm, when "the patient, who had flattered himself with pleasing hopes from his apparent health on the intermediate days, would now find them all dashed by the gloomy change of his disorder." As the epidemic acquired force, and finally assumed the entire mastery, the remittent disappeared.²

The early epidemics of this city furnished frequent examples of the complication under consideration. If we make allowances for the peculiar language of Dr. Rush—the result of his medical theories—we shall have no difficulty in finding the proofs of this in his writings. In 1793, cases occurred which commenced like an intermittent or remittent, and ended with the usual symptoms of yellow fever. In others, the tertian type discovered itself after the more violent symptoms of the fever had been subdued (iii. 72). Similar occurrences were noticed in 1794, 1797, and subsequent years. In 1802, the fevers "generally *blended* their symptoms in their different stages.

¹ Fever of Wilmington in 1802, p. 20.

² Drysdale, Med. Mus., i. 41, 370.

The yellow fever often came on in the mild form of an intermittent, and even a febricula; and, as often, after a single paroxysm, ended in a mild remittent or chronic fever. When it appeared in the latter form, it was frequently attended with a slow or low pulse, and a vomiting and hiccup, such as attended in the yellow fever" (iii. 213, iv. 15, 78). Speaking of 1803, Dr. Rush says: "In every part of the city it often came on, as in the year 1802, in all the milder forms of autumnal fever formerly enumerated, and went off with all the usual symptoms of yellow fever. Again, it came on with all the force and malignity of a yellow fever, and terminated in a day or two, in a common remittent or intermittent. These modes of attack were so common, that it was impossible to tell what the character, or probable issue of a fever would be for two or three days (iv. 86).

In like manner, Dr. Currie, in his account of the epidemic of 1793, states that the usual disease of the season, the remitting fever, was often blended with this disease. This, at least, he thought not improbable—remarking, in accordance with his peculiar etiological views, that though two epidemics arising from a change or alteration in the sensible qualities of the air, or from any invisible miasma diffused abroad to a considerable extent in the atmosphere, cannot exist together; yet, there is nothing more certain than that a contagious disease, and a disease depending on climate, season, and soil, may exist at the same time and in the same place (p. 32.)

In another work he reverts to the subject, stating that the complication occurred in numerous instances in the aforesaid year—the yellow fever assuming the remittent type.¹

In the later visitations of the fever in this city, cases of that kind did not frequently occur; for the localities where the yellow fever prevails are no longer the haunts of remittents and intermittents, and the disease, therefore, retains its purity of character. When modified cases do occur, they are found in persons who have taken the disease in the infected district, but reside in, or frequently visit, the suburbs or some other malarial district, and there imbibe the seed of some form of periodic fever.

That the cases of modified disease alluded to are the effect of the process mentioned—a complication of two separate and independent fevers—the engrafting of yellow on autumnal fevers, or the reverse—and should not be viewed in the light of instances of a different form of one and the same complaint, is a point upon which there can be but little room for dissent, or at least, which must be admitted to be worthy of serious consideration as more consonant with the principles of a sound pathology, and better harmonizing with facts we daily witness, and too glaring to be denied. Let us not forget that even by those who refuse their assent to the independent character and specific nature of the yellow fever, and regard it as merely a different form—the most aggravated—of common paludal fever; as also by some who though, while discarding the doctrine of the unity of fevers, so far as regards those mentioned, admit that the yellow fever may vary in type without suffer-

¹ *Bilious Fever*, p. 11.

ing a modification in any of its essential attributes, and are skeptical in relation to the coexistence of two febrile poisons in the production of the case to which the attention of the reader has just been called; let us not forget, I repeat, that by all these the fact is well known, and by some of them recorded, that the yellow fever, like other diseases of the same class, imparts, as we have seen, when it reigns epidemically, its livery to all other complaints. Now, all this livery wearing amounts to nothing more than the amalgamation of some of the phenomena appertaining to the yellow fever with those characterizing the disease thus modified; and as, in order that the former should exhibit themselves in conjunction with the latter, the cause producing the one set must produce a morbid impress on the system at the same time, or soon after, or before that producing the other, it follows that we have in all instances of the kind, examples of complication. If the reality of this process be admitted in regard to those instances, there can be no reason to deny that a similar result may obtain relative to cases in which symptoms of yellow fever show themselves in conjunction with those of autumnal fever, or the reverse, in which the former absorbs the latter, to a greater or less extent.

Nor must we lose sight of the fact that the modifications which form the subject of our present remarks, are never encountered except in places and localities where paludal or autumnal fevers prevail to a greater or less extent, or among individuals who have been exposed a sufficient time to the atmosphere of such places to imbibe the seed of the diseases accruing therefrom. The morbid cause floating in that atmosphere produces, when in its simple or uncomplicated state, autumnal fever, and the cause floating in yellow fever localities when equally uncombined, produces yellow fever; but the former never gives rise to yellow fever, or the latter to common autumnal fever. When, therefore, we find cases presenting phenomena appertaining to both diseases, or when we find one supplanting the other, in persons who have been exposed to both causes, or in places where both diseases prevail simultaneously, we may infer that those cases are the offspring of the co-operative action of both causes, and consequently the effect of a complication of two diseases.

We have seen that complications of yellow fever with other diseases are not unfrequently encountered. No one has denied their existence so far as concerns yellow fever and dengue, dysentery, rheumatism, &c., and when at New Orleans, in 1832, and perhaps elsewhere, the symptoms of the fever were combined with, and superseded by those of cholera, I am not aware that any one there, or anywhere else, affirmed that the assemblage of phenomena produced, constituted a mere form of yellow fever or cholera, and was the result of a single cause. No one refused to admit that it was the product of a combination of two causes, which, when acting separately, produce yellow fever on the one hand, and cholera on the other; and hence, that it should be regarded in the light of a complication. If we admit the explanation in these cases, we shall have some difficulty in discovering the propriety of rejecting it in instances in which the phenomena, superadded to those of yellow fever,

are those of autumnal fever instead of cholera, &c., or when the disease supplanted by the former, happens to be a remittent or intermittent, or some other complaint.

Yellow fever, in its pure and uncontaminated state, never any more presents the symptoms characterizing the anomalous cases in question than it does those of acute rheumatism, dengue, dysentery, or cholera Asiaticus, and if, in patients exhibiting the latter, we recognize the existence of something superadded to the phenomena of the yellow fever itself, and therefore produced by a separate cause, the same holds with respect to the something added to, or which preceded, the symptoms of autumnal fevers. In both instances, extraneous phenomena are superadded to those of yellow fever—in both instances, two sets of causes must have been at work to produce the effect observed; in a word, in both instances, we have before us examples of the complication of two distinct diseases, and not a peculiar form of either.

The occurrence of the amalgamation of the morbid poisons giving rise to yellow and bilious remittent fevers presents nothing repugnant to the principles of a sound pathology, and is not incompatible with the laws of the economy. Experience has long shown the fallacy of the Hunterian maxim, which teaches that no two different fevers can exist in the same constitution¹—a maxim from which Barthez started, in establishing his doctrine of the distraction of forces,² and about which so much has been said. It has been shown that though, in most cases, one of those diseases acquires the supremacy over the other, and either expels it or keeps it in check, yet, in others, they progress simultaneously; that this takes place even in relation to eruptive fevers, a fact denied by Hunter; and that the whole of what has been remarked in the preceding pages is conformable to certain laws which should not be overlooked by those who undertake to describe the true and fundamental characters of diseases, and to assign to these their nosological position. When we investigate the subject as it undoubtedly deserves to be investigated, we find that intercurrent, endemic, or even epidemic diseases, are not only influenced by the meteoric constitution of the period at which they appear, but that their characters, phenomenal and anatomical, are constituted at the outset of those appertaining to them, and also, to a greater or less extent, of those transmitted by complaints arising from anterior medical constitutions or reigning causes; and toward the close of their prevalence, of those characterizing succeeding complaints. We find that such diseases seldom manifest themselves in their pure and unmodified garb except towards the middle of their epidemic career, when the influence of anterior and of succeeding constitutions or morbid causes is not felt; that what takes place in the same locality during different periods occurs at the same epochs in different localities—diseases which retain their purity of character in particular places, losing it as they reach other places where different morbid influences prevail; and that much the same results

¹ On the Blood, introd., 4th ed., p. 3; Works, by Palmer, iii. 3.

² *Nouv. Elem. de la Sc. de l'homme*, ii. 181.

obtain in regard to diseases which attack individuals exposed to other morbid influences elsewhere, or in the same place at other seasons.¹ The reader is already aware that Sydenham long ago taught that the epidemics of autumn often insinuate some of their symptoms into the winter diseases which follow. All know that Dr. Rush had noted the complications thus produced. He recorded their occurrence in his account of our earlier epidemics, and well observed that "it belongs to powerful epidemics to be followed by similar diseases after they disappear, as well as to run into others at their first appearance" (iii. 101).

Be this, however, as it may, malarial or autumnal bilious fevers form combinations with very many diseases, and might, *à priori*, easily be understood, in the absence of positive proofs, to coexist with the yellow fever in the way described. The combination of periodic fever with pneumonia and pleurisy is too well known to need my enlarging upon it in this place. The bilious pleurisy of some writers, and of the public at large, as well, indeed, as every instance of periodic pneumonic inflammation we hear of in our southern States, and elsewhere, are examples in point—complications of pneumonic inflammation with autumnal fever.² Malarial fevers have been known to combine with scarlatina anginosa,³ typhus, typhoid, dengue, catarrh, influenza, dysentery, smallpox, scarlatina, measles, erysipelas, syphilis, scabies, whooping-cough, oriental plague, cholera, &c. The relapsing fever of Ireland, in 1847, was not unfrequently complicated with pneumonic inflammation. The plague is sometimes combined with the same disease, with smallpox and other eruptive complaints, quinsies, dysentery, and cholera. Typhus and typhoid fevers combine with smallpox, measles, erysipelas, and other eruptive diseases, cholera, &c. The petechial fever has been known in association with gout, colic, and intercurrent diseases generally. The sweating fever prevailed epidemically in the department of Dordogne, in France, during the greater part of the year 1841, affecting 10,803 individuals in a population of 83,342, and causing a mortality of 797, or one in about 5.5 of the sick. For two years previous, the department was overrun with eruptive fevers—measles, scarlatina, smallpox, and varioloid. Generally, one or two of these occupied the ground for three or four weeks, and then made way for the others. In several instances, measles and scarlatina, or smallpox and varioloid marched together; while, in some, all four existed at the same time. Under these circumstances, the sweating fever made its appearance. In the course of the year—from the 7th of May to November—the disease broke out five times in as many different places, and disappeared; so that the epidemic, considered in its *ensemble*, appeared, as it were, in five instalments. On four of these occasions, it assumed suddenly its legitimate garb, and immediately drove away all other diseases.

On the fifth, however, things took a different turn. All at once, after a

¹ See on these subjects Boudin's *Géographie Médicale*, p. 22, and Fuster's very excellent work, *Des Maladies de la France dans leurs rapports avec les saisons*, pp. 193–199.

² *Pneumonia and Malaria*, p. 437.

³ Rush, ii. 245.

thunderstorm, on the 7th of May, and the days following, a disease heretofore unknown broke out. It was very different, in its nature and results, from the measles, which then prevailed; but yet difficult at first to recognize, owing to its being under the dependence of the complaint it so singularly and suddenly came to replace. The disease no longer presented exactly the same train of phenomena, the same kind of pulse, or eruption. Nevertheless, there was in that new physiognomy of symptoms a family air which greatly puzzled the physicians. The measles were modified. To the precursory febrile symptoms were added others which are strangers to the disease; while others which belong to it disappeared unexpectedly. Some days before, the patients had been seized with a chill, cough, coryza, and all the opening symptoms of measles. In some cases, one of these symptoms now gave way; in others, a different one disappeared; in all, the attack henceforward came on without the chill. Some, however, had a marked and well-defined rubeolic eruption; while in others the skin became covered with a miliary eruption.¹ To this interesting fact may be added another, somewhat akin to it, derived from high authorities. Storck and Lepecque de la Cloture mention epidemics of miliary fever during which the pneumonia which prevailed terminated, not by expectoration, but by miliary eruptions, more or less abundant, or by fetid sweats.

Similar combinations exist in relation to common miliary fever. Dengue unites itself with scarlatina; influenza with the same disease, measles or bilious fever. The readers of Dr. Rush cannot have forgotten a passage in which, in describing the influenza which appeared in the summer of 1807, at the same time, in all the States of the Union, he observes: "The bilious fever which prevailed in August imparted to it several of its symptoms. There were obvious remissions and intermissions, great pain in the back, and apparent cessation of the symptoms of the disease on the third, and a return of them on the fourth day. The disease appeared, in one respect, to be a monster, *its head and breast wore the character of influenza, while its trunk and limbs indicated it to be a bilious fever.*"²

All these things have been observed, not once, but often; and, surely, when we find physicians of respectability detailing such complications, and have seen some of them ourselves; when we are told, by no less an authority than Dr. Macbride, that "the measles, next to the smallpox, is the species of eruptive fever most commonly met with—as was observed, particularly, in the Foundling Hospital of Dublin in 1760," and that "a number of the children having been inoculated for the smallpox, in the meanwhile were seized with the measles, and both species of eruption were perfectly distinct;"³ when we are told of this complication, examples of which have been seen in this city and in many other places; when we find that smallpox has existed, in the same system, in

¹ Parrot, *Histoire de la Suette Miliairé*; *Mém. de l'Acad. de Méd.*, x. 395, 396.

² *Works*, iv. 101. See Baker (Sir G.), *Treatise, &c.*, in *Collection of the Sydenham Society*, p. 73; Holland, *Med. Notes—Connection of Certain Diseases*, p. 64, Am. ed.

³ *A Methodical Introduction to the Theory and Practice of Physic*, p. 376.

association with scarlatina, hooping-cough, the vaccine poison, and syphilis; that scarlatina and measles, or scarlatina and hooping-cough, occasionally combine together; that typhus exists sometimes with erysipelas, scarlatina, vaccinia, psora, syphilis, or gonorrhœa; that plague has been known to exist in combination with smallpox, vaccinia, syphilis, or cholera Asiaticus; dengue with scarlatina; influenza with the latter disease, or measles; syphilis with herpes, or psora—we may well pause before we express a doubt as to the propriety of the explanation of the mixed phenomena in the cases alluded to. The instances of complication here mentioned must be sufficient for every unprejudiced mind; and surely when, in addition, we are informed of three distinct diseases, as smallpox, measles, and hooping-cough, or measles, scarlatina, and chickenpox, running their course simultaneously; when we are told that inoculation with a mixture of variolous and vaccine matters will produce, not, as Woodville stated, one or other of the two diseases, but *both*; when a case is cited, on the authority of Leroux, in which the vaccine pustule was, as it were, imbedded in the variolous, and the matter of each, when used for inoculation, produced its specific disease; when, I say, we are credibly informed of such occurrences—shall we be justified in doubting the accuracy of those observations, and in maintaining that, instead of assemblages of distinct diseases, we have in all such instances really and substantially but one complaint, modified somewhat by peculiarities of season and weather, or other causes, but still, to all intents and purposes, the same? And can we, in the presence of such facts, refuse to acknowledge that the same explanation is applicable to cases in which the symptoms of yellow fever coexist with those of autumnal fever, as well when the former disease has succeeded in supplanting the latter completely, or in great measure, as when periodic paroxysms survive the other disease?¹

¹ See, on these various combinations, Nott, N. O. Journ., March, 1848, p. 586; Dickson, Trans. of Med. Assoc., v. 142, 143; Williams on Morbid Poisons, i. 40, 120, 211, 212, 264, 301, ii. 38, 65, 191, 296, 623; Holland, Connection of Diseases, p. 58—Am. ed., p. 64; Blair, p. 70; Heberden, p. 385; Fodéré, Méd. Légale, v. 352–357; Lafont-Gouzi, *Materiaux pour servir à l'Hist. de la Médecine Militaire, &c.*, pp. 47–83; Sarcone, Mal. de Naples, ii. 225; Anglada, *Traité de la Contagion*, i. 331–336; Adams on Morbid Poisons, pp. 11–13; Bousquet, *Traité de la Vaccine*, p. 300; Robertson, A General View of the Nat. Hist. of the Atmosphere, ii. 370; Ettmuller, p. 321; Du Pré, Charleston Journ., v. 607, 608; Lachaise, Bulletin de l'Acad. de Méd., xiv. 1043; Bonnet, *ib.*, xv. 79, 80; G. Sutton, Rep. to Indiana State Med. Soc., p. 62; Huxham on Fevers, p. 61; Bailly, F. Int., p. 253; Gouraud, F. I., pp. 93, 103; Rienzi, *Miasmi Paludosi*, p. 121; and others referred to by the present writer, in his work on Pneumonia.

CHAPTER XXII.

DURATION, CONVALESCENCE, RELAPSE.

DURATION.—From what has been said in the foregoing description of the yellow fever, the reader must already have formed some idea of the duration of the disease. He, therefore, scarcely need be told, that in the large majority of cases it runs its course and ends in death or recovery in a short time. But although the fact may have been foreseen, it is one of sufficient importance and interest to justify a few words on the subject. Dr. Currie, in his account of the epidemic of 1793 (p. 28), states, that in fatal cases the disease ended on the fourth, fifth, or sixth day—sometimes earlier, sometimes later; and that, when recovery took place, it usually did so before the fifth day; frequently on the second or third day—sometimes at an earlier period. “When the patient survived the seventh day without the accession of black vomiting, coma, or scorbutic symptoms, or great or increasing debility, the disease generally terminated favourably” (p. 29). Again: in a later publication, after he had witnessed several epidemics, Dr. Currie remarked, that when the disease terminated favourably, it was mostly between the third and the seventh day; when the reverse, it was on the fourth, sixth, or eighth day.¹ Dr. Rush, also (iii. 73, 4), found that, in 1793, the fever ended from the third to the eleventh day. It dragged on, in some cases to fifteen, twenty, or even thirty days. But such cases, which also appeared in subsequent seasons, were not of frequent occurrence. On the other hand, in 1797, he saw cases which terminated in death in twenty-four hours (iv. 13). Dr. Barnwell, in like manner, remarks, that at first either death or a perfect recovery took place in a few days, and that many died on the third or fourth day (p. 376). Deveze, another of our high authorities, found that death not unfrequently occurred within three days from the outset of the attack—sometimes in a few hours (pp. 27, 28). In 1805, according to Dr. Caldwell, death generally took place—sometimes in the course of the second day from the commencement of the second stage, and on the fifth or sixth day from the commencement of the attack. At other times, but seldom, the disease ran its course more rapidly, death taking place on the third or fourth day, or in thirty-six or twenty-four hours; while, again, it was occasionally prolonged to two weeks from the outset of the symptoms (pp. 85, 86, 87). In a subsequent work, the same physician states, that the duration varies from three to seven days. In 1820, according to Dr. Jackson (p. 59), the disease ended usually, in fatal cases, in three, four, or five days; while in some it extended to the seventh or eighth day. Recovery, in some instances, took place after

¹ On Bilious Fever, Appendix, p. 220.

a paroxysm of a few hours or days; while, in more severe ones, it was seldom retarded beyond the eighth.¹

It would appear from the foregoing statements, which are corroborated by that of Nassy (p. 20), and the observations made in 1853 and 1854, that in this city, at various epidemic periods, death from the yellow fever has most frequently occurred on the fourth, fifth, and sixth day—that not unfrequently it has occurred on the third day, or thereabout; that, in some cases, the disease has run its course to a fatal termination in a much shorter period—in thirty-six or twenty-four, or even sometimes in a few hours; while, in some instances, on the contrary, the patient has survived to the eleventh, fourteenth, or even later. It results, also, from the same statements that recovery in mild cases often takes place early—at the close of the first or inflammatory stage, from twenty-four to seventy-two hours—that in others it is postponed to from the third to the seventh day; that when the disease continues beyond the ninth or fourteenth day, a circumstance of unusual occurrence, it rarely proves fatal; and that though cases present themselves which linger on to the fifteenth, twentieth, or even thirtieth day; such cases, the last particularly, are but seldom encountered, and must be regarded as constituting, like those of shorter duration than three days, exceptions to a general rule; and that, finally, whether the disease terminates in recovery or in death, it runs its course most usually in from three to nine days.

The fever in question differs in nothing, so far as regards the rapidity of its course, in the large majority of cases, and the limits of time within which it terminates in recovery or death, from that of other places. If here, instances are found in which it ends within the first three days, or in less than forty-eight or thirty-six hours; if it occasionally proves suddenly fatal, the occurrence of like instances has, over and over again, been recorded in other cities of the United States from the earliest prevalence of the disease to the present day.² Such also will be found to have been the case in the South of Spain and in Italy,³ as well as in various parts of hot climates.⁴

Labat, in his quaint way, says: “A convenient circumstance connected with this disease was, that it carried off people without loss of time. In six or seven days, at most, the affair was terminated.”—*Ce que cette maladie*

¹ The reader will not forget what was mentioned in the early part of this volume (p. 52, &c.), that in 1699, the disease ran its course in seven or eight days, but that some were “well and dead in four days.”

² Lining, ii. 426; Monson, p. 181; Baxter, xxi. 3; Gros, p. 13; Thomas, p. 89; Dickson, Phil. Med. and Phys. Journ., iii. 256.

³ Berthe, p. 79; Arejula, p. 161; Pariset, Fev. of Cadiz, p. 32; Velasquez, p. 12; Jackson, Fev. of Spain, pp. 45, 54; Pym, p. 60; Ib., Edinb. Journ., xxxv. 41; T. Smith, xxxv. 42; Rochoux, pp. 519, 568; Palloni, p. 6; Ib., Ed. Journ., ii. 68; Dufour, iv. 54.

⁴ Pinkard, iii. 112, 114–15; Warren, p. 16; Poissonnière, p. 50; Caillot, pp. 22, 23; Bally, p. 272; Gilbert, pp. 66, 73; Moreau de Jonnes, p. 138; Chisholm, i. 194; Moseley, p. 440; Pugnet, p. 351; Imray, liii. 82; R. Jackson, Sketch, i. 92; Birnie, xiii. 333; Ralph, ii. 42; Guyon, Réponse, p. 28; Rufz, p. 55; Levacher, p. 73; Bancroft, p. 35; Wilson, p. 9; Walker, Med. Rep., i. 496.

avait de commode, c'est qu'elle emportait les gens en fort peu de tems; six ou sept jours tout au plus terminaient l'affaire.—“It has happened to some persons,” he says, “who only suffered from a slight headache, to fall dead in the street” (i. 73).

In more modern times, Moreau de Jonnes has said: “Une cruelle expérience ne laisse point douteux qu'aux Indes occidentales il n'y ait des malades qui soient frappés de mort à l'instant même de l'apparition des premiers symptômes” (p. 138).

If, in this city, the termination on or about the third day, is of more frequent occurrence, we find a like period assigned as the common duration of the fever elsewhere.¹

If, again, in a large number—indeed, the majority—of cases, the termination of the fever in this city takes place between that day and the ninth, inclusive—more frequently about or between the fifth and seventh; similar results have been obtained wherever the yellow fever has prevailed—the limits assigned varying somewhat within these two periods, according to the several authorities, viz: 3—5; 3—6; 3—7; 3—8; 4—5; 4—6; 4—7; 4—9; 5—6; 5—7; 5—9; 6—7; 6—8, &c.²

If, in addition, the disease among us is occasionally, but in a limited number of cases, prolonged to, or beyond the ninth day, to the eleventh, fourteenth, or even the twentieth, thirtieth, or thirty-sixth day, like protractions have not uncommonly been noticed elsewhere.³

¹ Monson, p. 181; E. H. Smith, 127; Cartwright, p. 11; Palloni, p. 6; Ib., Edinb. Journ., ii. 88; R. Jackson, p. 127; Pym, xxxv. 41; Ib., Bulam F., p. 60; Arejula, p. 161; Amiel, Edinb. Journ., xxxv. 268; Dufour, iv. 54; Audouard, p. 55; Du Tertre, i. 423; Levacher, p. 73; Lempriere, ii. 66; Imray, liii. 82; Ralph, ii. 69; Birnie, xiii. 333; Gilbert, pp. 66, 88; Pugnet, p. 358; Desportes, i. 197; Bancroft, p. 15; Dariste, p. 138; J. Clarke, p. 16; Dyott, p. 1005; McArthur, p. 348; Arnold, p. 249.

² Valentin, p. 173; Thomas, pp. 85, 89; Gros, pp. 10, 12; Rufz, p. 15; Barrington, xii. 325; C. Drake, xxi. 125; E. H. Smith, p. 127; Monson, p. 181; N. O., 1820, p. 10; Cartwright, ix. 11; Baxter, xxi. 3; Ticknor, iii. 229; Irvine, p. 31; Tully, p. 301; Dickson, iii. 256; N. O. 1839, p. 326; Humboldt, p. 776; Joubert, p. 969; Leblond, pp. 105, 7; Audouard, pp. 54, 5; Dufour, iv. 54; Arejula, p. 161; Pym, 60; Ib., Edinb. Journ., xxxv. 41; Amiel, ib., pp. 267, 8; Velasquez, p. 12; R. Jackson, *Fev. of Spain*, p. 127; Palloni, p. 6; Pariset, p. 462; T. Smith, Edinb. Journ., xxxv. 42; Walker, *Med. Repos.*, i. 496; McArthur, p. 348; Dyott, p. 1005; Dariste, p. 138; Maher, p. 877; J. Clark, p. 16; Lempriere, ii. 88; Bancroft, p. 15; Vatable, pp. 346, 7; Gilbert, pp. 66, 73; Pugnet, p. 358; Birnie, Edinb. Journ., xiii. 333; Ralph, p. 69; Imray, liii. 82; Levacher, p. 73; Catel, p. 13; Madrid, pp. 6, 7; Blair, p. 74; Gillespie, pp. 45, 6; Bally, pp. 232, 255, 272; Rouppe, p. 301; Savarésy, p. 267; Chisholm, i. 194; *Dict. des Sci. Méd.*, xv. 362; Caillot, p. 23; Rochoux (F. J.), p. 89; R. Jackson, *Sketch*, i. 75, 88.

³ Barrington, xii. 325; Irvine, p. 31; N. O. 1839, p. 326; Dickson, iii. 256; Tully, p. 301; Baxter, xxi. 3; Cartwright, ix. 11; Monson, p. 181; N. O. 1820, p. 10; E. H. Smith, p. 127; T. Smith, *Ed. Journ.*, xxxv. 42; Velasquez, p. 12; R. Jackson, *Fev. of Spain*, p. 127; Arejula, p. 161; Dufour, iv. 54; Rochoux, pp. 519, 568; Pariset (1819), p. 32; Palloni, p. 6; Ib., *Ed. Journ.*, ii. 88; Chisholm, i. 192; Rochoux (F. J.), p. 89; Bally, p. 232; Gillespie, pp. 45, 46; Levacher, p. 73; Pugnet, p. 358; Gilbert, p. 73; Bancroft, p. 16; Dyott, p. 1005; H. McLean, p. 94; Walker, *Med. Repos.*, i. 496; R. Jackson, *Sketch*, i. 75, 88, 110.

Lastly, if recoveries from our yellow fever often occur in from twenty-four hours to three days; if other recoveries take place from the fourth to the seventh, eighth (generally three, five, seven), and if the few cases which are protracted to a later period, seldom end fatally; experience has shown that such is also the case elsewhere.¹ "Cases," says Blair (p. 61), "have been admitted into the Seamen's Hospital, and private cases have been noticed, death-stricken from the first, wherein the system seems to have been so saturated with the poison as to have prevented any stage of excitement." The same writer remarks, in reference to the fever of Demerara in 1837: "The average duration of an attack of an epidemic ending in convalescence, estimated from 1158 cases of the gravior form, and 428 cases of the mitior form, was 6.34 days for the former, and 5.35 for the latter. The average duration of a fatal attack, estimated from 404 cases, was 7.08. In these estimates, the day of attack and the day of death or convalescence, are reckoned each one day. Although the maxima and minima days of duration differed widely from this average, those of the gravior being 23 and 2, and those of the mitior 13 and 2, and those of the deaths 24 and 2; yet the vast majority of cases fell in closely with the averages, and this coincidence happened always more particularly when the disease was purest and the epidemic pulsations most intense. The averages may, therefore be assumed as the law of duration of each class respectively" (p. 74).

From the foregoing statement it results, that in whatever part the true yellow fever has made its appearance, its duration has differed from a comparatively few hours to several days—varying, in the large majority of instances, from the third to the ninth. The latitude is somewhat extensive, and the frequency of the fatal or favourable issue on the several days embraced within the period mentioned—the third, fifth, seventh, ninth, or even the fourth, sixth, and eighth, seems at variance with the opinion of those who regard the yellow fever as invariably a disease of seven days.² That death or recovery may often take place on that day; that such may have been the case in a particular manner during the epidemic of St. Domingo in 1802, as mentioned by Bally (p. 272)—though neither Gilbert (pp. 73–4) nor other writers on the disease, affirm anything of the sort—may, perhaps, be true; but it is evident that, generally, whether here or elsewhere, matters assume a different turn—so frequently, indeed, that we would, from that fact alone, be justified in suspecting that some, at least, of the advocates of the opinion in question had in view rather a malignant remittent than the true yellow fever.

As may be presumed, the duration of the disease varies somewhat—though not to a very great extent—not only in different places, but also in the same

¹ N. O., 1839, p. 228; Pariset (1819), p. 32; Pym, xxxv. 41; Smith, xxxv. 42; Tully, p. 301; Madrid, pt. 1, pp. 6, 7; Vatable, p. 346; Amiel, in Johnson, p. 268; Monson, p. 181; Lining, ii. 416; Arejula, p. 161; E. H. Smith, p. 127; Rufz, p. 15; Baxter, xxi. 3; Rochoux (F. J.), p. 89; Ib., *Fièvre Amaril*, pp. 344, 519; Chisholm, i. 192; Irvine, p. 31; Caillot, p. 19.

² *Dict. des. Sci. Méd.*, xv. 362; Hastings, p. 39; Humboldt, p. 776; Tully, p. 302.

places—at different epidemic seasons. Cases in which no reaction takes place, and the patient sinks at once, or in a short space of time; or in which, though characterized by reaction, the disease runs through its various stages to a fatal issue in a very rapid manner, do not show themselves with equal frequency everywhere. Experience shows that, though not unusually encountered in this country, Spain, and Italy, they are more common in tropical regions, as pointed out by Moreau de Jonnes (p. 138), Caillot (p. 23), &c. They are not encountered as often in some epidemics as in others. They are mentioned as not uncommon in this city in 1793 by Rush, Deveze, &c. In 1794, Dr. Rush saw or heard of no case which terminated in death on the first or third day. In 1797, on the contrary, they were encountered. While such was the fact here, and while rapid cases were seen in New Haven by Monson, they are not noticed at other times. Dickson states that, in 1817, the disease ended on the fifth or sixth (iii. 206). In New Orleans, in 1820, it did so from the third to the fifth (p. 10). Humboldt says that, at Vera Cruz, it usually extends beyond the sixth or seventh day (p. 776). On one occasion, mentioned by McArthur, it ended fatally from the second to the fourth; on another, from the third to the fifth (p. 348). Catel (p. 13) says that, at Martinique, the fever commonly ended within the first seven days. Rufz (p. 15) rarely saw it end before the fourth, or after the seventh. Rouppe says its duration was generally four, or at latest five days (p. 301). Levacher, from seven to fourteen (p. 73). Savarésy, from four to five days, or five to six (p. 267). Pym, four or five days, or fifty hours (p. 61). Rochoux found it varying in the West Indies from four to eight days; at Barcelona from two days, or a few hours, to thirty-six days (p. 568).

The termination also varies as much at different periods of the same epidemic, being generally shorter at the commencement than at a more advanced part of the season. In reference to the epidemic of 1793, Dr. Rush says: "I think I have observed the fever to terminate on the third day more frequently in August, and during the first ten days in September, than it did after the weather became cool (iii. 7). Dr. Barnwell (p. 376) points out the same tendency to a more rapid termination at the outset and before the weather became cooler. Dr. E. H. Smith (p. 127) made the same remark in relation to New York in 1795; and Dr. Irvine says, in relation to the epidemic of Charleston in 1817: "At first the cases terminated on the fourth day. As the season advanced, they extended to the sixth day. Towards the end of summer, they were sometimes protracted to the eighth or thirteenth" (p. 31). In like manner, we are told by Dr. Anderson, of Mobile, that the cases occurring late during the sickly season of 1853 seemed to run a longer course than those which occurred in the height of the epidemic. They were not less malignant than their predecessors.¹

The duration of the cases varies according to the different forms assumed by the disease—a circumstance on which the variations previously mentioned in great measure depend—the rapidity being proportionate to the violence of the

¹ Trans of Med. Assoc. of Alabama (1854), p. 49.

attack. Bancroft, who does not seem to have seen malignant or congestive yellow fever, remarks that the disease, in its most violent form, destroyed the patient on the third or fourth day, or sooner. "When," he says, "it assumes its mild form, death usually happens between the seventh and fiftenth day" (pp. 15, 16). Similar remarks are made by Cartwright (p. 11), Gilbert (p. 73), Savarésy (p. 267). In the first and second forms described by the latter, the disease ended on the fourth, or during the fifth day. In the third form, it ended on the fifth, sixth, or seventh; and, in the fourth form, on the sixth, seventh, eighth, sometimes ninth day (283-4).

In the second form described by Ralph, the scene generally closed on the fourth day (p. 67). The third form was milder and more protracted, extending to the fifth and seventh; sometimes, but more rarely, to the ninth day (p. 71). In the fourth, or insidious and congestive form, the patient died on the first, second, third, or fourth day (p. 72). Dr. R. Jackson also points out these differences, stating, that in the concentrated inflammatory species, the disease seldom extends beyond the fifth day. In the mild or mitigated inflammatory, it is often protracted to the seventh, ninth, or fourteenth day (p. 59). In the mild gangrenous, the progress to death is often rapid, sometimes complete, within the fourth or fifth, but sometimes extends to the tenth day, or a fortnight (p. 71). The concentrated gangrenous sometimes ends before the close of the first day, but more frequently terminates on the third or fifth day (p. 74), while the mild phlegmatic form was sometimes long protracted, running to the twenty-first day (p. 87); and the concentrated form of that species proving fatal, sometimes early—by convulsion or coma—in many cases not until the seventh or the fourteenth day, and even later (p. 91).

It remains to be mentioned in reference to this matter, that the manner in which the law of duration is disturbed, is, as Dr. Blair has correctly stated, by an abnormal prolongation of some one of the stages, or by the interval between the stages being so prolonged as to amount almost to convalescence, or suddenly cut short by resolution, or fatal congestion, or nervous shock. The report of Dr. Wragg, so frequently referred to, furnishes a fit illustration of the disturbing influence in question. In 201 of the cases treated by him in the Roper Hospital, the duration of the first stage varied from eight hours to seven days, distributed as follows: 1 case, eight hours; 2, twelve hours; 12, twenty-four hours; 3, thirty-six hours; 52, two days; 102, three days; 24, four days; 2, five days; 2, six days; and 1, seven days. Similar observations, with regard to the second stage, showed the following results in 155 cases: In 1 case it lasted fifteen hours; in 80, twenty-four hours; in 2, thirty-six hours; in 67, two days; in 4, three days; and in 1, four days.¹ In these enumerations, we have at once a key to the variableness of duration of the disease, which, as a necessity, must be influenced by the prolongation or abbreviation of either of these stages, or both, if they happen to combine in the same case. What Dr. Blair remarks relative to the fever of Demerara will, to a considerable extent, apply to occurrences here and elsewhere. Healthy activity

¹ Charleston Journ., x. 70, 71.

of the secretions, such as bilious stools and copious urine indicate, have often a tendency to prolong the duration of fatal cases. Treatment has an effect on the law of duration; and complications, and secondary symptoms, and relapses from ill-managed or imperfect convalescence, also disturb it (p. 74).

CONVALESCENCE.—In cases of recovery, the period of time which elapses before the patient regains his pristine condition of health—in other words, the length of the state of convalescence—as well as the modifications of functions, organic and animal, and the morbid changes in the organs and tissues which present themselves during the progress towards a complete restoration, vary in no small degree. On this, as indeed on every other point connected with the disease, the most contradictory statements and opinions will be found recorded. By one set of writers on the yellow fever of both tropical and temperate climates, convalescence is said to be generally, if not always, rapid and unimpeded. We learn, for example, from Dr. Barton, that, at New Orleans in 1833, “it was not found necessary, in a single instance, to resort to tonics during convalescence, which was usually very rapid, and just in proportion to the preceding unembarrassed state of the constitution.” When there was no chronic ailment, “the return of appetite and strength was inconceivably rapid.” To the same effect, in relation to the fever of this country and Europe, and the West Indies, are the asseverations of several respectable and high authorities.¹

On the other hand, we have the authority of many writers for the fact that convalescence, though generally short, is sometimes slow and tedious. Examples of the kind have been noticed in the fever of this city. There are not wanting those who contend that, so far as their observations extend, such is the usual character of the period in question. Pym, who, as we have seen, represents the convalescence as progressing rapidly in the fever of Europe, found it, in a particular instance at least, slow in the West Indies (p. 13). In severe cases, according to Louis, convalescence (in Gibraltar) was generally long—that is, in proportion to the duration of the disease, the mean extent of which was seven or eight days. The strength was not perfectly re-established sooner than in from ten to twenty days. Similar results occurred in mild cases, in which the duration of the convalescence was long in proportion to that of the disease. In these, indeed, the disproportion was found even greater than in severe cases (pp. 173–176). According to Dickson (iii. 257), Gros (p. 14), Townsend (p. 174), C. Drake (xxi. 136), and Girardin (p. 36), in this country; Rochoux (p. 518), Amiel (xxxv. 280), Velasquez (p. 17), and Palloni (p. 4), in Europe; and Caillot (p. 169), Gilbert (p. 67), Steward (*Fev. of Grenada, Regist.*, iii. 187), Maher (p. 863), Pugnet (p. 374), Adair (in *Caisergues*, p. 126), Monro (*Report*, quoted by Gillkrest, ii. 272), Imray (liii. 93), Bally (pp.

¹ Merrill, ii. 237, ix. 255; Copland, iii. 143; Jourdain, v. 260; Veitch, p. 132; Blair, p. 89; Chervin, Report on Catel, p. 16; Cartwright, ix. 11; E. H. Smith, p. 127; Kelly, xiv. 373; A. Smith, p. 501; Pym, pp. 248, 249; Robert, ii. 703; Pariset, p. 437; Ralph, ii. 78; Musgrave, ix. 135; Vatable, p. 350; Dickinson, p. 40; Rufz, p. 53; Osgood, p. 15; Rochoux (F. J.), pp. 163, 164; Ib., Rech. sur les Différentes Maladies, &c., p. 344.

268-272), Madrid (p. 27), and the reporters on the Fever of Cayenne in 1850 (p. 170), in tropical climates, it is more frequently slow and tedious than otherwise. The diagrams in which Dr. Blair has exhibited the day when perfect recovery took place after convalescence—*i. e.* the length of the latter—show that, in the whole number, including the mild and severe forms, convalescence ended at some period between the first day after the termination of the disease and the twenty-first; in the greater number, on the sixth, next on the fifth and seventh. In the gravior form, the duration varied from one to twenty-four days; the greater number being on the sixth, next on the eighth, seventh, and ninth day. In the mitior, convalescence was more uniform in its duration, taking place in six, four, three, and five days; next in seven, eight, and two; then in nine, ten, and eleven. The average number of days during which patients convalesce from the yellow fever remained in the hospital till perfect health was restored was, for 417 mitior cases, 6.55; and for 1,140 gravior cases, 7.91. Retarding and accelerating causes acted with as much force on this period as on that of actual disease (p. 74).

This diversity of statements finds an echo in the result of inquiries into the fever of this city, convalescence from which is said by some to have generally proved long and tedious, or painful;¹ and by others, as generally rapid and secure. That instances of the former kind have been encountered among us, and frequently too, no one who is conversant with the disease, or who will consult our authorities,² will deny. Dr. Rush remarks that, in some cases, convalescence was very slow, and that he was more than usually struck by the great resemblance which the system in this state bore to the condition of the body and mind in old age. A like statement has been made elsewhere. But, though such cases of slow recovery are undeniably met with, in greater or less number, at every visitation, it may safely be said that, in this city, at least, convalescence from attacks of the yellow fever is, in a large proportion of instances, rapid, and, with due care on the part of the physician and patient, secure. Of this, indeed, there can be no doubt: that convalescence from this fever contrasts advantageously with that from the remittent and bilious fevers of various grades, in which recovery is usually protracted.³ So evident is this difference, among us at least, that it may be, and has been adduced, with great show of plausibility, as a point of distinction between those two forms of fever.

As may be readily presumed, the length of convalescence will be found to differ according to a variety of circumstances—the external influences to which the patient is exposed; the peculiar character of the attack; its duration and the degree of its severity; the previous state of health, and particular constitution and temperament of the individual; and the mode of treatment pursued. As a general rule, it has been found—so, at least, we are told—that convalescence is more rapid in sporadic attacks of

¹ Deveze, p. 33.

² Rush, iii. 159; Monges, ii. 58; Chapman, ix. 130; Caldwell (1826), p. 167; Klapp, Recorder, iv. 85.

³ Musgrave, ix. 135; Vatable, p. 350; Monges, ii. 58; Klapp, iv. 85.

the yellow fever, or when the epidemic constitution is one of limited force, than when the disease is the result of a wide-spreading cause; owing, doubtless, to the difference in the nature and character of the attacks occurring under these diversified circumstances. In the same way, doubtless, we may account for the fact of the length of convalescence differing in various epidemics. Sir William Pym found it slow in the West Indies, and rapid, some years after, at Gibraltar (pp. 13, 248). Amiel, Louis, and others, found it slow in the latter place during the epidemic of 1828. Rochoux¹ represents it as being rapid at Guadaloupe, and usually slow and tedious in Barcelona. Catel (p. 16) and Rufz (p. 13) found it rapid at Martinique in 1838-9; Ralph (ii. 78) in Barbadoes; Musgrave (ix. 135) in Antigua; Vatable in Guadaloupe, &c. Now, a perusal of these writers will show that the character of the diseases they describe varied in no small degree—being inflammatory in some instances, malignant and congestive in others. That such is the true explanation of the discrepancy in question, we may infer from the fact that, during the same epidemic, a difference as regards the speed and completeness of convalescence is found to occur, according as the attack has been of an inflammatory or of a congestive character—long, and embracing the several stages of the fever; or short, and consisting only of the early stage. Dr. Wilson (p. 38) called attention pointedly to the fact that in the inflammatory form convalescence is generally rapid and complete, while in the congestive it will often be slow and precarious; and similar observations have been made elsewhere. In mild attacks, and especially when the disease does not progress beyond the first or inflammatory stage—and these cases constitute most of those that end in recovery—the convalescence is usually rapid, sometimes extraordinarily so.² It is much less so when the patient experiences some of the severe symptoms of the next stage; while, in more protracted cases, it is commonly slow and tedious, often attended with incommoding, unpleasant, and even dangerous phenomena, owing to “the amount of repair which is necessary to restore the dilapidated organs,”³ to reinvigorate the nervous system prostrated by the morbid poison, and to replenish the bloodvessels with a pure and healthy fluid.

It has usually been found that the rapidity of convalescence is proportionate to the unembarrassed state of the constitution at the time of the attack. Dr. Barton, in his account of the epidemic of New Orleans in 1833 (p. 19), properly dwells on this fact; and what Dr. Ralph says of the results obtained in Barbadoes in 1817, will be found to apply with equal propriety to those obtained everywhere else: “Convalescence in this fever was generally remarkably rapid, and recovery perfect, when the constitution, previous to the attack, had been sound, and the patient young, which was the case with most of the Queen’s [Regiment]. On the contrary, those whose habits were very drunken, or whose constitutions were unsound, had their health much injured by the fever” (ii. 78). Nor is

¹ Rech. sur la F. J., pp. 163, 164, 344; Ib., Rech. sur Différentes Maladies, p. 518.

² Kelly, xiv. 373; McArthur, pp. 352, 353; Madrid, p. 28; Audouard, p. 60; Pariset, p. 437.

³ Wood, i. 299.

it less certain that much will depend, in respect to the length and completeness of convalescence, on the remedies pursued for the cure of the disease, as well as on the period at which the case has come under medical treatment. Hence, we find it stated, in reference to our yellow fever, that proper treatment, aided by a judicious regulation of hygienic means, seldom fails to render the convalescence secure and rapid.¹ Sir William Pym (p. 13) states that slow convalescence was principally found to occur, in the West Indies, in the few who had survived venesection; and the experience of this city and country, where, as we shall see, the lancet has been used in an indiscriminate manner, goes far to lead us to presume that such may very readily have been the result during the deadly epidemic Sir W. noticed at Martinique, and where the copious loss of blood may have prostrated the system beyond the point of speedy restoration. To subject a patient, in any disease, to considerable depletion when his power of reaction is but feeble; to stimulate him, and place him under the use of tonics, when his case is of an inflammatory character; or, again, to make him undergo a perturbative medication when, perchance, he requires repose and the simple action of the restorative powers of nature; or by any means to cause him to remain with partially cured organs—is not only to endanger his life, but also to subject him to the risk of remaining feeble long after his recovery, or with organs and tissues in an unfit condition to perform healthfully their functions, and, as a natural consequence, to entail on him a protracted convalescence. If such is found to be the case in diseases generally, we can easily foresee that a like result will obtain in the yellow fever, the effect of the poison of which upon the system is already too prostrating and perturbative to need being rendered more so by adventitious means. Nor is it less true that, in order to insure a prompt convalescence, the disease must not be allowed to take too firm a hold of the system before being subjected to proper medical treatment. Dr. R. Jackson, whose authority cannot be appealed to too often, remarks, as the result of his long experience, that if every person is “put under medical care at an early stage of indisposition—that is, within twelve hours or less from the time of attack—and if suitable means of remedy be then applied, under a full comprehension of the principle on which effect depends, the course of the disease may be speedily cut short in most cases, convalescence may be rendered rapid, and the recovery of health, for the most part, perfect.” In the contrary case, if time was lost at the commencement, “the course of the disease was not cut short, and the recovery was not complete; in other words, the disease ran its course, and therein laid the foundation of chronic ailments, the removal of which was often a tedious and difficult task, sometimes an impracticable one.”²

The phenomena manifested during protracted convalescence vary in different individuals:—

A. In some, the case is characterized by general, but undefined indisposition, with or without protracted jaundice.³

¹ Caldwell (1826), p. 167.

² Sketch, ii. 204, 205.

³ Girardin, p. 37; Merrill, ii. 237.

B. Most of those in whom the yellowness of the skin is long in fading, as well as in a large number of others in whom it disappears rapidly, or has prevailed slightly or not at all, labour long under debility of the whole system, or simply of the limbs or joints.¹

C. Some are affected with œdematous swellings of the feet, ankles, and legs, and even with ascites.²

D. The patient is not unfrequently troubled with vague and long-continued pain in the limbs, as well as in the loins and head.³

E. The pulse often remains full and slow.⁴

F. In some cases, the senses are blunted; the mind being often uncommonly depressed, and the memory impaired, with also a great aptitude to shed tears.⁵

G. In other cases, the patient is troubled with vertigo, bewilderment, and even delirium.⁶

H. Not unfrequently the patient suffers from complete pervigilium, or short continued, and disturbed sleep.⁷

I. In many instances, the convalescent is much troubled by the formation of large abscesses on the body and limbs, and of obstinate and unhealthy sores or pustules, furuncles, and buboes, which do not suppurate kindly, and are often very difficult to heal.⁸

K. It is not unusual to find the nervous system under some of the aforementioned circumstances in a high state of excitability, with great impressionability to the action of cold, and a marked tendency to the production in some climates of tetanic symptoms.⁹

L. In many cases, the leading symptom is a protracted and obstinate susceptibility and irritability and nervous irritation of the stomach, characterized by the usual effects of that morbid state—sense of fulness, pain, nausea, vomiting, &c.¹⁰

M. In other instances, the gastric symptoms appear to be the result of torpidity of action of the stomach, and manifest themselves under the garb of flatulency, loss of appetite, impaired or slow digestion, &c.¹¹

N. In a few cases, on the contrary, as Dr. Rush (p. 159) has noticed, the appetite becomes inordinate, and is with difficulty controlled.

¹ Rush, iii. 159; Pariset, p. 437; E. H. Smith, p. 127; Velasquez, p. 17; Townsend, p. 174; Imray, liii. 85; Pariset, p. 438; Rochoux, p. 518; Fever of Cayenne, in 1850, p. 170.

² E. H. Smith, p. 127; Arejula, p. 216; Resueno (Burnett, p. 235).

³ Maher, p. 868; Fever of Cayenne, p. 170; Joubert, p. 976.

⁴ Pariset, p. 439.

⁵ Rush, iii. 159; Girardin, p. 37; Bally, p. 272; Pariset, p. 438.

⁶ Rochoux, p. 550.

⁷ Rush, iii. 159; Girardin, p. 37; Pariset, p. 438.

⁸ Dickson, iii. 257; Wood, i. 299; Resueno, in Burnett, p. 236; Fever of Cayenne, 1850, p. 170.

⁹ Girardin, p. 37.

¹⁰ Rochoux, p. 518; Velasquez, p. 17; Girardin, p. 36; Pariset, pp. 438, 9.

¹¹ Amiel, xxxv. 280; Bally, p. 272; Girardin, p. 36.

o. Nor are the abdominal functions less often implicated. In some cases, the bowels are torpid or costive—more frequently they are affected with obstinate, or even chronic diarrhœa, of a serous or mucous character.¹

p. Not a few of the worst cases of the kind exhibit considerable emaciation, which leads to a state of cachexia and marasmus.²

q. Among the phenomena enumerated during long protracted convalescence, may be noticed loss of the hair of the head, or its being changed suddenly to a gray colour.³

r. I have already stated that convalescence from this fever is marked in some instances by a sudden revival of the venereal appetite.

s. In some cases, the patient is affected with cough and other pulmonary symptoms, which are sometimes difficult to relieve or cure, and even assume a phthisical character.⁴

t. Combined with some of the preceding symptoms, we sometimes notice a dry, parched skin, small, quick pulse, a griped appearance of the physiognomy, and other symptoms of slow fever. These cases enter within the category of those which Dr. Rush, in his account of the epidemic of 1797, denominated *chronic*⁵ (iv. 15).

As to the pathological conditions of the tissues and organs in cases attended with the phenomena above enumerated—debility, œdema, impaired gastro-intestinal functions, &c., a difference of opinion has existed and continues to exist. That many instances may occur in which these effects are the results of simple chronic inflammatory or nervous irritation of the mucous tissues of some portions of the alimentary tube with secondary derangement of the larger and important glands, and that these morbid states may, after some time, be removed, or end in death without producing disorganization of the parts implicated we may readily assume. At the same time, however, there is nothing in the nature of the yellow fever calculated to lead to the denial, even were we destitute of positive facts revealed by autopsic investigation, and even did not analogy lead us to the conclusion, that in some cases at least they may be the consequence of chronic obstructions of the viscera. Sir William Pym, it is true, affirms that, out of many hundred cases of the Bulam fever which he saw, he never met with one chronic affection of the liver; and appeals, in confirmation of this, to the return of sick at Gibraltar for January, 1805, and for December, 1813, '14—pointing out this fact as an additional proof of the difference between that fever and the bilious remittent which seems to be connected with a derangement of the system, and a common sequel of which is a chronic affection of the liver (p. 224). Copland states the same fact, and reaches a similar conclusion (iii. 143). Catel informs us (p. 16) that of more than a thousand patients treated in the hospital of which he had charge at Martinique, not one left with symptoms denoting the existence of a morbid

¹ Bally, pp. 272, 568; Girardin, p. 37; Joubert, p. 976.

² Girardin, p. 37; Bally, p. 272.

³ Rush, p. 159; Resueno, in Burnett, p. 236.

⁴ Bally, p. 272; Arejula, p. 216.

⁵ Fever of Cayenne, 1850, p. 170.

state likely to become chronic. The records of yellow fever, in this and other cities, contain but few references to chronic organic affections. Dr. Musgrave also says, that visceral obstructions, as a consequence, as far as he has yet been able to ascertain, have been extremely rare, or even unheard of (ix 135). To a certain extent, we may safely admit the inaptitude of the disease being followed by such sequelæ, not only for the reason assigned by Dr. Bancroft—the concentration, virulence, and instantaneous action of the miasma, and the acute attack and rapid course and termination of the fever resulting therefrom; but also for the reason, that the cause does not, as we shall have occasion to see, necessarily produce its effect on the viscera in question.¹ But, while admitting this, and recognizing that in cases wherein such affections remain, they may be the effects of fortuitous complications, experience points out the fallacy of denying the possibility of their occurrence in cases free from such complications. Thus, we find Arejula (p. 216) stating, that ascites, or general dropsy, and sometimes, but less frequently, hectic fever and pulmonary consumption supervened as effects of the disease. Risueno, too, remarks that “in many instances, the disease terminates in ascites, or some other species of dropsy”²—the frequent effect of visceral obstruction. We learn from the official report of Mr. Monroe on the fever as it occurred in the 77th regiment at Falmouth, Jamaica, in 1827, that “in cases of recovery from this form of the disease, the patient was generally a considerable time afterwards affected with some organic complaint of the lungs, liver, spleen, or other viscera; and Dr. Alfonzo de Maria has recorded that, after a Cadiz epidemic, several, who had passed through the disease, were recommended to go to San Lucar in consequence of the visceral diseases which followed.”³ In the epidemics of this city, instances of the kind have occasionally been noticed. Dr. Charles Drake, of New York, informs us that convalescence from the aggravated cases in the epidemics of that city, in 1819, was remarkably tedious, there appearing to be a serious lesion of some important organ, from which the patients recovered with difficulty.⁴

RELAPSES.—Convalescence being once fairly established—be the attack one of serious or of mild character—the question presents itself whether individuals who have gone through the disease and recovered, are liable to be again affected with the same train of phenomena before reacquiring their pristine health; in other words, whether they incur the risk, not of a second attack, but of a relapse. The question was long ago agitated in this city and elsewhere, and has been the subject of contradictory statements. By some it is maintained, 1st, that such an event never or seldom happens; the yellow fever, in that respect, assuming the course of many other diseases produced by specific poisons, in which the characteristic symptoms, when once removed, never show themselves so soon after the attack as to constitute a regular relapse; and 2d, that fever, if it presents itself at such a period, is, except in very rare instances, of a different character from the true dis-

¹ Sequel, p. 96.

² See Burnett, p. 235.

³ Gillkrest, Cyclop. of Pract. Med., ii. 272.

⁴ Med. Repos., xxi. 136, 137.

ease; or if otherwise—if the attending symptoms are those of the yellow fever, properly so called—there is equal reason to doubt the genuineness of the first attack.

We are told by Lempriere, that in the continued endemic of the West Indies—a form of fever identical with our yellow fever—he never knew an instance of a patient falling into the same train or kind of symptoms after he had acquired the convalescent state; but when, he continues, “from inattention, or any other cause, a relapse threatened, it never failed to put on the form of a common intermittent, and yield to the administration of the bark; in the remittent endemic, relapses are more frequent, and they usually bring the remittent form again” (ii. 68). Dr. Musgrave says: “We never met with what could strictly be called a relapse but once—it was caused by exposure to the sun, and strong drink, before strength was nearly recruited; it proved fatal, with black vomit, in twenty-four hours from the time of the second attack” (ix. 135). Dr. Veitch is still more positive: “Instances of relapsing were not known at the hospital at Antigua, particularly when they were received early, and an opportunity afforded of thoroughly subduing the disease by decisive evacuations.” In other instances, and when determinations to internal organs, of a chronic nature, had taken place, these often suffered relapses; but such relapses could not be called relapses—they were symptomatic” (p. 109).

Rufz saw no case of relapse at Martinique in 1838 and 1839 (p. 53). Dr. Dickinson says they are “almost impossible” (p. 40). Pym states, that while relapses are frequent in the bilious remittent, he cannot call to his recollection one relapse in the Bulam, or true yellow fever (p. 225). Pariset also observed at Barcelona, in 1821, that when the disease was not of the worst kind, and convalescence was rapid, relapses never occurred, unless as the result of great imprudence in diet; and even in such cases, the consecutive disease seldom assumed the form of yellow fever (p. 437).¹ In this country, Dr. Cartwright, of Natchez, remarks that, when convalescence once took place, it was never interrupted by relapses. To this rule he has seen no exception; though he admits that Dr. Gustine, in whose judgment he placed great confidence, *thinks* he saw a few.² Dr. Kelly admits that the paroxysm of the fever, when it does not end in collapse and death, or in convalescence, may leave the organism in a state of susceptibility that renders it peculiarly liable to a recurrence of fever. “This consecutive fever is the result of the derangement of the secretory functions, of visceral obstruction, or chronic inflammation of the tissues, the sequelæ of previous violent action. But this secondary disease cannot be adduced as an illustration of the occurrence of relapses, for the symptoms produced are not those of yellow fever.”³ Dr. Dickson tells us that at Charleston relapses are rare, or rather unknown (p. 352). Dr. Ashbel Smith affirms, that relapses, properly so called, do not occur. However, “patients, imperfectly or seemingly convalescent, do, by impru-

¹ Copland, iii. 143; Craigie, Edinb. J., lx. 416; Dickinson, p. 40.

² Med. Recorder, ix. 15.

³ Am. Med. J., xiv. N. S., p. 373-4.

dence in eating, or exposure, fall into the last stage, or that of prostration, characterized by black vomit, hemorrhage," &c. Or it may happen that, during the stage of metaptosis, the patient may walk about, eat, &c., and then have black vomit and die. But, in such cases, the vascular excitement or paroxysm is not renewed, and hence the case cannot be called a relapse, for the reason that in all such instances, "the hemorrhagic state of the system subsists during all the time of the delusive pause."¹

On the other hand, not a few writers speak of relapses as of more or less frequent occurrence, affirming that they appear at all periods during the course of the convalescence, and that the symptoms of the second are, in every respect, similar to those of the primary attack, the disease being, in that respect, governed by the same laws as other complaints arising from kindred causes. They are mentioned, so far as regards the yellow fever of hot climates, by Wilson (p. 38), Vatable (p. 349), McArthur (p. 354), Osgood (p. 15), Caillot (p. 15), Gilbert (p. 67), Ralph (ii. 70), Imray, *Ed. J.*, (liii. 85), Ib. (lxiv. 328), Maher (p. 883), Boyle (215). "When febrile action has ceased," says Dr. Robert Jackson, "the advances of health often proceed in a regular and favourable course for a given time. The disease reappears at a certain point, either precisely similar, or differently modified in appearance from the preceding (ii. 211)." "Relapses," he adds, "which occur under a system of repletion, are numerous—sometimes serious—the symptoms conspicuous in the organs of the abdominal cavity more than in other parts of the system" (ii. 313).

Dr. Blair states that relapses and exacerbations, even after the characteristic symptoms of the severer form (the gravior), were not rare at Demerara, but generally occurred close after the period of convalescence. He cites thirty-one cases of relapses or exacerbation (p. 86).

In the fever of Cadiz, in 1800, according to Sir James Fellowes—apparently on the authority of Arejula: "Relapses were very frequent and fatal; for, when the patient found himself getting better, he perhaps walked out, and not having strength, this treacherous disease carried him off suddenly" (p. 63). "Relapses," according to Roehoux (p. 518), "are easily induced in the Amaril typhus of temperate regions." Louis, in speaking of the mild cases observed at Gibraltar, in 1828, says: "A relapse was rare; it took place where the disease had been violent, and then almost all the symptoms observed the first time reappeared—at least it was so in a great many cases" (p. 176). Amiel frequently noticed similar cases at Gibraltar in 1814 (270). The fever described by Drs. Denmark² and Boyd³ as occurring in Sicily, may not be the true yellow fever, but it certainly approximates to it in many of its features. In it relapses were not unfrequent. But, laying the example of this fever aside, we may, in addition to the authorities cited, adduce those of Gillkrest (p. 280), and Baneroff (*Sequel*, p. 93), the latter of whom appeals to the testimony not only of several physicians already

¹ Tr. of N. Y. Acad., i. 59.

² Med.-Chir. Trans., vi. 313.

³ Johnson on Tropical Climates, p. 301.

mentioned, but also of Drs. Gray, Dickson, and Mr. Tiber, of the Navy, and Messrs. Lea, Wald, Martindale, of the garrison of Gibraltar, of Dr. McArthur, Messrs. Sproale, Short, Humphrey, Thomson, and Sutherland—several of whom are also adduced, for the same purpose, by Sir William Burnett (pp. 482-4).

In our own country, relapses in yellow fever have been alleged to be of not unfrequent occurrence, and have been observed in most places where the disease has appeared epidemically.¹ In a case mentioned by Dr. Ticknor, the disease broke out anew after the patient had been convalescent two days. It was brought on by imprudent exposure, and terminated fatally in two days. No black vomit was ejected, but the symptoms presented were not to be mistaken, and the matter was found in the stomach after death.²

Dr. Fenner remarks that there was one feature in the epidemic of New Orleans in 1853 which is worthy of mention—that is, *a tendency to relapse*, which presented itself in many instances. “After the customary subsidence of the fever, at the end of the third day, there would be a period of calmness lasting from twenty-four to forty-eight hours, and then the fever would kindle up again, and last for one day or more. This secondary fever was often very dangerous, though it frequently terminated happily. The interval of convalescence, after the first attack, was sometimes so long as to appear more like a second attack than a relapse. From two to four weeks have been known to elapse between two distinct attacks of the fever in the same person. Heretofore these relapses, or second attacks, were not at all common. On the 4th of November I was called on board a steamboat to see a gentleman who had just come down from the village of Warrenton, situated on the river, just below Vicksburg, where the yellow fever had been very bad. He said this was the fourth attack, or relapse, he had suffered since July; he died of black vomit on the 8th” (p. 53).

From what precedes, and a survey of all that has been said on the subject, we might be prepared to conclude that relapses in the yellow fever, though observed at different times and in different places, cannot be viewed as occurrences frequently encountered. To maintain that they never do, or that they cannot occur, would be to oppose the asseverations of authorities of too high a character to be doubted, and to nullify the results of observations made here. A relapse may indeed take place at any time during convalescence, when the poison has not been completely eliminated from the system, or when a perfect cure has not been obtained. After that period, the recurrence ceases to be classed among relapses, and assumes the character of a second attack. But, at the same time, it would perhaps be equally hazardous to regard them as common results, seeing, as we do, that they are either denied by some, or viewed as of limited occurrence—as exceptions to a general rule—by many of those who entertain less exclusive views; while it will be found that those who describe

¹ New Orleans 1819, p. 9; Thomas, p. 87; N. O. 1820, p. 7; Baxter, xxi. 3; Perlee, iii. 16; Cartwright, ix. 15.

² N. A. Med. and Surg. J., iii. 231.

them as presenting themselves frequently, are mostly to be met with among the supporters of the identity of the yellow with the common remittent fever, and who therefore may be suspected of having regarded as relapses of the former what were strictly relapses of the latter, or the occurrence of an attack of yellow fever in an individual recovering from remittent fever, and *vice versâ*. It is not less probable, also, that, in some instances, what has been denominated relapse was, strictly speaking, a second attack. Even in the quotation I have made from Dr. Fenner, we may perceive a confusion of ideas in reference to this matter, relapse and second attack being mixed up together as one and the same thing. According to Dr. Fenner's own showing and acknowledgment, many of the instances referred to under the head of the latter sort were of the former, the individuals having recovered from the first attack when again seized with symptoms of yellow fever. Nor are we certain that the paroxysm of fever which came on after the period of calmness—no usual phenomenon in true yellow fever—bore always the character of the disease; and as to the case from Warrenton, whether we regard the attacks in one light or another, I presume Dr. F. will not feel disposed to vouch for the accuracy of diagnosis of the individual who gave him the information. To this let it be added, that relapses, or the recurrence of the symptoms of true yellow fever, after convalescence from an attack has set in, are oftener met with in individuals who go through the disease and remain in infected localities, than in those who, having imbibed the seeds of the fever there, sicken elsewhere. Under the former of these circumstances, the return of the disease after recovery may properly be regarded as the result of reinfection, inasmuch as the individual continues exposed to the influence of the febrile poison, and his system is in a less favourable condition to resist its impression when the latter is brought into play through the agency of some exciting cause.

To these opinions I am the more inclined, as they receive support from the experience obtained in this city. For here relapses, though occurring in some instances, are not frequently met with¹—convalescence, in cases of recovery, progressing with more or less rapidity to a restoration of health; or, if interrupted, being usually replaced by symptoms appertaining to pathological states different from those of yellow fever. Whether this infrequency of relapses should be ascribed to some peculiarity in the nature of the disease; to the specific nature of the cause; to the apprehensions and actual weakness of convalescents, which renders them very cautious in avoiding exciting causes; or to the efficacy of the means employed—I shall not attempt to decide positively: but shall content myself with the remark that, though unwilling to refer the effect solely to the first of these causes, or to refuse acknowledging some influence in the latter, it is impossible to deny that much, perhaps the greatest share of the result, is to be ascribed to something peculiar to the disease, inasmuch as an almost equal unfrequency of true relapses—compared with what takes place in kindred febrile complaints—occurs where no extra caution is observed on the part of convalescents, as

¹ Caldwell (1805), p. 79.

well as in cases subjected to opposite modes of treatment. But, be this as it may, that by proper caution and appropriate treatment a decided influence is frequently obtained, experience amply shows; for, though sometimes occurring from the action of some unappreciable agency, relapses are generally the effect of improper conduct on the part of the patient—muscular exertion, exposure to cold, unsuitable diet, indulgence in the venereal act, or the like; while it has been observed, over and over again—here and elsewhere—that in yellow fever, as indeed in most other diseases, relapses are less generally met with in cases in which the disease has been properly eradicated, or treated in a way calculated, while husbanding the powers of life, to leave the organs devoid of morbid irritation, as well as in individuals subjected during convalescence to particular modes of treatment. In regard to the latter, Dr. Robert Jackson, who was an advocate of energetic means, remarks: “The English practitioners generally adhere to the rule of full living; the French and most other practitioners enjoin a rigid abstinence during convalescence. They even enjoin laxatives at frequent intervals, cooling drinks, and simple food, peremptorily interdicting strong wines or other strong liquors. Relapses are less frequent under such management than under the preceding, but recovery is slow” (ii. 213).

Louis (p. 176) found at Gibraltar, in 1828, that relapses rarely occurred in cases of a mild character, but took place where the disease had been violent. The reverse has often been observed in this country¹ and Europe; and we may even say, on good authority,² during the very epidemic described by the French pathologist. As might be expected, they occur more frequently at an early than at an advanced period of convalescence; for in the latter the organs and tissues have regained a large share of their pristine vigour, and may the better resist the action of morbid causes. It has generally been found that relapses are highly dangerous, and there are not wanting writers who affirm that they usually, or even almost always, lead to a fatal issue.³ But, though this may be true as regards a large number of cases, I am disposed, from what I have seen, to join in sentiment with those who maintain that, however dangerous they may be, the danger to be apprehended from them has not unfrequently been unduly exaggerated; for cases often present themselves, in which a relapse has done little more than retard recovery.⁴ In general, the danger diminishes in proportion to the length of convalescence.

As is well known, the late Dr. Robert Jackson was of opinion that relapses are principally to be expected at septenary periods—the seventh, fourteenth, twenty-first, and twenty-eighth day—with new and full moon.⁵ That such a disposition to the recurrence of disease at those stated periods manifests itself in some forms of fever, few will be disposed to deny. It is found to occur in intermittent fever, and there are not wanting facts to show that the tendency to relapse in remittent fever is governed by the same laws that regu-

¹ New Orleans in 1820, p. 7.

² Smith, xxxv. 42.

³ Pariset, p. 457; T. Smith, xxxv. 42; Baxter, xxi. 3; Gilbert, p. 67; Fellowes, p. 63; Ralph, ii. 90; Imray, liii. 85; Wilson, p. 38; Vatable, p. 349; McArthur, p. 354.

⁴ Thomas, p. 87.

⁵ Sketch, ii. 212; Outline, p. 304.

late the process of incubation, periodic decrease, and exacerbation of symptoms and critical efforts. We know that in that form of fever which, from its constant tendency to return at stated times after apparent convalescence, has received the name of the relapsing fever, the symptoms, after the critical sweat of the fifth or seventh day, generally return on the fourteenth; and that those relapses recur not once only, but several times. Other fevers, in like manner, show a disposition to recur at stated periods. In the government of Ufa (Russia), autumnal fever, which in that section of country is very common, attacks the patient every seventh day only, and is so severe that it generally proves fatal.¹ Similar to what occurs in relation to the decline of the disease, or to its attack after exposure to the cause, the periods most remarkable, according to the ample experience of Dr. R. Jackson, for the recurrence of the symptoms, are the septenary. Whether the same will be found to hold in regard to yellow fever, further observation must decide. If so, the *tendency* alone, not the law, is applicable only to cases in which the occurrence takes place spontaneously; for it is plain that relapses occasioned by exciting causes must occur at any time these are applied, though perhaps more especially at the septenary periods.

CHAPTER XXIII.

PROGNOSIS.

GENERALLY speaking, the prognosis in the yellow fever is unfavourable; for, under ordinary circumstances, and except in some epidemic seasons of unusual mildness, the disease is of the most dangerous character, and the chances of recovery are slender. The difficulty of arriving at anything like certainty on the subject, and the impropriety of being positive as regards the opinion to be expressed in reference to the result, is enhanced, as Towne noticed more than a century ago, by "the velocity with which nature arrives at the happy or fatal period" (p. 61), the sudden and unexpected changes which often take place in the condition of the patient, and the little aid afforded in it by various signs which in other diseases are of the highest value. No one acquainted with this form of fever will refuse his assent to the statement of Arejula (p. 449) and others, that cases occur in which physicians of experience and practical tact have thought a patient free from danger when almost immediate death belied the prediction; while, on the contrary, cases of recovery occur when, from the nature of the symptoms, a very different result might naturally have been anticipated. Nor could it be otherwise. The yellow fever presents this peculiarity, that while in other disorders a favourable issue may be looked for from the healthful continuance of certain important func-

¹ Notices of Russia, United Service Journal, Jan. 1833, p. 49.

tions, or the return of these to their normal state, in the former these functions may continue apparently undisturbed, or vary but little from their state of health, and nevertheless the disease march with certainty to a fatal termination. "From the frequency of the pulse," says a writer of this country, "the appearance of the tongue, the temperature of the surface, or clearness of the intellectual faculties, a favourable prognosis should be formed with extreme caution, as these frequently do not vary from the standard of health in cases of extreme danger."¹

But, however this may be, as the disease is not invariably fatal—and, indeed, as in some seasons it occasions but a limited mortality—it is necessary to discern and point out such phenomena and signs—for such there must be—as will enable the physician to arrive at as near an estimate as possible of the probable issue of the case. For the purpose of attaining this knowledge it is necessary, as the reader may infer from what precedes, to discard all reliance on any single symptom or circumstance of the case; for the condition of any of these—the particular state of any of the excretions and secretions, the morbid change in any organ or function—cannot be of much avail in furnishing the desired information, unless we take into consideration at the same time the modifications which have taken place in all the other leading, and even in some of the secondary symptoms. In a word, it is only from an attention to the general state of the patient, or the result of a combination of all the signs, that we can expect to form an idea of what the event is likely to be.²

1. We shall have occasion to see, in the forthcoming chapters of this work, that the disease is more or less modified as to the degree of its prevalence, the severity of its attack, and the mortality it occasions, by certain peculiarities connected with the condition and habits of the patient, his age, sex, race, constitution, idiosyncrasy, &c. Such being the case, it follows as a necessary consequence, that when it occurs under the circumstances the least favourable in the above respects, the prognosis will be equally unfavourable. Hence, we need not so much fear the issue in children, females, negroes, or those who have already passed through the ordeal of the disease, in natives of the warmer latitudes, in those whose habits are temperate; while youth, a plethoric state, a sanguine constitution, high living, and intemperance, predispose to a debauch, excessive fatigue, or terror, a fit of anger, the intemperate use of venery, &c., often excite a severe attack, from which the chances of recovery are less to be anticipated.³ "The attack," says a distinguished physician, "is apt to be violent, and its progress hasty, in the sanguineous and plethoric. For the intemperate, there is almost no hope." "The Irish, Germans, and Scotch, afford us the worst cases. Spaniards, Italians, and Frenchmen, are very apt to recover. Midway stands the Englishman, the northerner, and the mountaineer, or inhabitants of our interior country. Generally speaking, the more recently a stranger has

¹ A. Smith (of Galveston), p. 32.

² Chisholm, i. 190.

³ H. McLean, pp. 97–8; Rush, iv. 33; Devezé, p. 30.

come here (Charleston, S. C.), the more severe the attack. Among the young children assailed, the ravages of this pestilence are very great."¹ The same may be said of the remote cause itself, which, in some seasons, is of such a degree of malignancy as to produce a disease, which, though apparently differing little from that of other periods, has a greater tendency to end fatally, and must, therefore, call for a very different prognosis. Again, the latter must be more guarded during the continuance of the same epidemic in different parts of an infected city—the symptoms appearing the same—inasmuch as the malignancy and fatal tendency of the fever differ in them. The same remark is applicable to the several periods of the same epidemic. In general, the prognosis should be more guarded at the outset, as the disease is more apt then to terminate fatally.

2. As a general rule, it may be said, that in yellow fever it is not so much the presence of good signs which we are to look for in order to form a favourable prognosis, as the absence of bad signs. For, what would be regarded as good signs in most other diseases, are of little or no avail in this, and many patients recover without having presented what may, strictly speaking, be called by that name; often after exhibiting some one or more of those which experience, in many fatal cases, teaches us to look upon with suspicion; whereas, the appearance of any one of the decidedly bad signs, and still more a combination of them, must be viewed with fear, leading, as they very generally do, to a fatal termination.

3. It is not less to be noted, that much more is to be expected from a gradual amendment of the febrile and other symptoms than from a sudden disappearance of even the most unfavourable among these. From the latter change, indeed, the most disastrous results may in general be expected—followed, as it generally is, by delirium, coma, and other bad symptoms.² The danger of the disease, great as it is, when the latter is uncomplicated with any other complaint, becomes much more so, and calls for a more unfavourable prognosis, when to the phenomena which reveal its existence, are superadded those indicating the coexistence of other disorders. (*Dariste*, p. 170.)

4. With the exception of the mildest or ephemeral forms of the disease, the danger to be apprehended is, generally speaking, proportionate to the shortness and rapidity of the case. When the disease extends to the seventh, ninth, or eleventh day, recovery may reasonably be expected. The same favourable view may be taken from the prolonged duration of the stage of reaction—the danger in the subsequent stage being proportioned to the shortness of the first and the early supervention of the state of metapostosis.³

¹ Dickson, *Essays Pathol. and Th.*, p. 353.

² *Dariste*, pp. 173–4; *Stone*, vi. 554; *Caillot*, pp. 162–3; *Chisholm*, i. 191; *Rochoux*, p. 547; *Currie*, p. 35; *Osgood*, p. 14.

³ *Lining*, ii. 428; *Deveze*, p. 31; *Dariste*, p. 174; *Dickson*, p. 354; *Caillot*, 161–5; *Copland*, iii. 148; *Pugnet*, p. 358; *Gilbert*, p. 66; *Bally*, p. 274; *Osgood*, p. 14; *Moultrie*, p. 13; *Ralph*, ii. 76; *O'Halloran*, p. 84; *Chisholm*, i. 191; *Rochoux*, pp. 516–547; *J. Clark*, p. 18.

5. In cases marked by remissions, the disease, as we have seen, is of a milder character, and admits, therefore, of a much more favourable prognosis, however severe the febrile reaction may be during the exacerbation.¹

6. Rigors, at the commencement of the attack, denote considerable danger, and are usually viewed as signs of fearful omen, the danger being proportionate to their duration.² The same may be said of chills, when violent, long continued, and repeated.³ By some practitioners the appearance of chills generally has not been viewed as a bad sign;⁴ by Arejula, slight chilliness was regarded as a grave one in the fever which prevailed at Malaga in 1802.⁵ Others, again, have represented the absence of chills as a sign of more serious import than the occurrence of rigors.⁶ In 1797, Dr. Rush, while regarding chills as indicative of a favourable issue, and affirming that the longer it continued the more favourably the disease would terminate, remarked that the first paroxysm coming on without any premonitory symptoms, or a chilly fit, was a sign of great danger (iv. 33).

7. *Jaundice*.—Considered in a general way, the yellow discoloration of the skin, which has given a name to the disease, is doubtless a sign of importance in a prognostic point of view; for, though not invariably observed in all fatal cases, it is much more frequently seen in these than in cases of recovery. Hence, generally speaking, the appearance of this symptom must be regarded as entitled to considerable attention, and held with suspicion. Much, however, in this respect will be found to depend on the period at which it comes on, and the hue or shade the skin assumes. Having already dwelt somewhat at large on this subject in a former chapter (p. 234–7), few words only will be required here.⁷

The appearance of jaundice at an early period of the disease may be viewed as a symptom of serious import, and as indicating a disease of dangerous, and even fatal character—the danger increasing in proportion to the deepness of the discoloration. When, on the contrary, it appears at a late period—after the sixth or seventh day—it loses the dangerous tendency in question, and, as we have seen, may even be said to assume the character of a critical sign. As regards the peculiar hue it presents: in many cases, and during certain epidemics, the light yellow or lemon colour has proved more dangerous than the dark yellow; while, in other seasons and localities, the reverse is said to have been the case. Jaundice is indicative of more danger when it assumes a greenish, violet, mahogany, or bronze hue, and particularly when the skin presents a mottled or party-coloured appearance, characterized by livid, olive, and ash-coloured patches of all sizes, and blending into each other. Greater apprehension is to be felt when the discoloration in ques-

¹ H. McLean, p. 102; Cycloped., ii. 274; Rush, iv. 32; Joubert, p. 972; S. Jackson, p. 61; Arnold, p. 17.

² Gillespie, p. 56; Fellowes, p. 61; J. Clark, p. 18.

³ Rush, iii. 52; Bally, p. 275; J. Clark, p. 18; Gros, p. 15; Gilbert, p. 78; Bone, p. 20.

⁴ Desportes, i. 197.

⁵ Arejula, Edinb. J., i. 450.

⁶ Kelly, xiv. 381; Fellowes, p. 61.

⁷ Rochoux, pp. 309, 553.

tion is rapidly and very extensively diffused over the body, than when it is limited in extent, and spreads slowly.

8. *Eyes*.—We have seen that redness of the eye is a common and even characteristic symptom of the yellow fever, and was of two kinds. In some instances it arises, as it were, from suffusion, the whole conjunctiva being equally red, and as if stained with blood; in others, it bears the character of an injection of minute and distinct vessels. Both these conditions furnish prognostic signs.

A. In general, it is found that considerable redness is a symptom of dangerous import, and that the apprehension it creates, increases in proportion to the extent of discoloration of the organ, which, at the same time, looks as if protruded.¹ A red eye, on the fourth or fifth day, especially when it becomes so after having been previously yellow, indicates great danger, and points to cerebral complications.

B. The glistening appearance of the eye, which arises from the minute, but distinct injection of the conjunctival capillaries; and the fiery and wild look, with which it is often associated, may almost always be regarded as a sign of great danger, indicating the probable occurrence of convulsions.²

C. Scarcely less dangerous, if not equally so, is the muddy or sombre hue and confused aspect of the organ.³

D. A fixed and steady stare—the eye remaining perfectly immovable—has usually been properly ranked among the most unfavourable signs, and is, indeed, the precursor of dissolution.⁴

E. On the other hand, considerable danger may be apprehended, when the conjunctiva acquires a pearly white colour—such as occurs in the worst cases of the congestive form of the disease.⁵

F. When the eyes assume a vacant and unmeaning expression, analogous to that of an idiot, the whole combined, or not, with the pearly whiteness before mentioned;⁶ when these organs are downcast, and remain half opened, sad, and bedewed with tears;⁷ or are hollow;⁸ or when their motion is sluggish, their aspect torpid, dull, inanimate and without expression—especially if they are at the same time injected and yellow;⁹ or when they have an expression of pensive sadness—of anguish and languor—betraying, at the same time, an inclination to shut out all objects, as if vitality were dormant or deficient;¹⁰

¹ Rush, iv. 34, 49; Rochoux, p. 296; Dariste, p. 172; Caldwell, p. 94; Caillot, p. 161; McArthur, p. 349; Gillespie, p. 56; Pariset, p. 449; Bally, p. 276; Jackson, p. 181; Kelly, xiv. 380; Lining, ii. 428; Gros, p. 16; Lind, p. 284; Seaman, p. 13; Harrison, ii. 321; Arnold, p. 18; Copland, iii. 148.

² Rush, iv. 34; R. Jackson (Spain), p. 120; Copland, iii. 148; Gros, p. 16; S. Jackson, p. 52.

³ Jackson, i. 181; Caldwell, p. 94.

⁴ Deveze, p. 30; S. Jackson, p. 52; Gros, p. 16.

⁵ R. Jackson, i. 182; Merrill, ix. 245.

⁶ Jackson, i. 182.

⁷ Dariste, p. 174; Bally, p. 276; Arnold, p. 23.

⁸ Currie, p. 37.

⁹ Jackson, i. 182.

¹⁰ H. McLean, p. 100; R. Jackson, i. 182.

or, again, when they roll about, "as if in search of something absent;" or agitated, as if by convulsive motions¹—danger is near at hand, and even imminent, and the issue doubtful.

G. Dilatation of the pupils, with red, protuberant eyes, is properly regarded in the same light, and is, under no circumstances—whatever may be the peculiar coloration of the conjunctiva—to be passed over unheeded, indicating, as it does, a condition of brain which may lead to coma, &c.² Nearly the same remarks are applicable to the undue contraction of the pupils.³

H. The continued increase of the redness afore-mentioned, instead of its diminution in the second stage, is of very serious import.⁴ Danger may also be apprehended when, after having subsided, this redness reappears at a subsequent period of the disease.⁵

I. The remarks made, relative to the jaundiced discoloration of the skin, apply equally well, in some particulars, to that of the conjunctiva, which is usually among the first, and often the only, part affected throughout the whole course of the disease. The prompt appearance of the yellow discoloration in the eye, furnishes the same prognostic indication, as the early diffusion of jaundice over the surface.⁶

K. Intolerance of light, and pain in the eyeballs, without inflammation, are unfavourable signs,⁷ indicating, as they do—especially when accompanied with rapid rolling of the organ—spasm, delirium, or coma.

L. When, on the other hand, the eye is calm and serene, bright at the onset—preserving an unclouded aspect, animated by hope, and undepressed by terror or apprehended danger—when the redness is not considerable and quickly disappears after the stage of reaction, and when withal the organ may be moved about with facility, we have the indications, if not of certain recovery, at least of a form of disease in which the chances are most in favour of the patient.⁸

9. *Face and Countenance*.—A high coloration of the face, its varnished appearance arising from a superabundant secretion of sebaceous humours, is a sign of dangerous import—the danger being in direct proportion to the degree which the redness attains.⁹ Dr. R. Jackson remarks that a florid tint, like Circassian bloom, is a suspicious symptom, particularly when it occurs at a late stage of the disease (i. 183). A pale and livid hue of the face and skin, generally in the early stage of the attack—connected as it is with the congestive and malignant form of the disease, is necessarily an unfavourable sign. The early appearance of a yellow suffusion, and at any period a sombre, leaden, violet, or greenish hue; or a dark mahogany or a clay or livid colour of the

¹ Jackson, i. 181–82; Gros, p. 16.

² Kelly, xiv. 384; Dariste, p. 172.

³ Dariste, p. 172.

⁴ Lining, ii. 430.

⁵ Rochoux, p. 297.

⁶ R. Jackson, i. 181; Rochoux, p. 297.

⁷ Jackson, i. 181–182; Lining, ii. 428; Gillkrest, ii. 273.

⁸ Rochoux, p. 297; H. McLean, p. 103; Jackson, i. 181; Gillkrest, ii. 273; Copland, iii. 148; Arnold, p. 18; Harrison, ii. 321.

⁹ Seaman, p. 13; Rochoux, p. 294; Cyclopaedia, ii. 273; Lining, ii. 428; R. Jackson, i. 183; *Ib.*, *Fev. of Spain*, p. 120; Gillespie, p. 56; Lind, p. 284.

face, will be found indicative of great or imminent danger; while the last may be viewed as certainly fatal.¹

When the countenance is agitated, or bears an expression of distress and anxiety, of sadness, of fear and despair, of moroseness and silent anguish, of sullenness or torpidity, of vacancy or gloomy indifference—when the features have the expression of laxity and suffering—when they are pinched (*grippés*)—when the eyebrows are contracted and impart a scowling, sinister look, with an expression of horror every time the stomach is pressed, or with *risus sardonius*, and twitchings about the mouth, the prognosis is most unfavourable.²

A swollen appearance of the whole face, with or without tension of the skin, or only of the nose and eyelids, as if bloated, indicates imminent danger;³ as does also a shrunken condition of the same part—which is observed in the later stages of some cases.⁴

On the contrary, when the countenance preserves its serenity, and presents, with the eyes, a steady and unclouded aspect—when the redness subsides at the period of remission—a happy issue may be anticipated.⁵ But, while the subsidence of this symptom must be hailed with satisfaction, we should be on our guard when we notice the sudden disappearance of the expression of agitation and distress already noticed, and a return of composure; unless, as has been remarked by a high authority,⁶ this be attended with a corresponding relaxation, expansion, and animation, since otherwise the tendency to torpor and congestion is declared, and the danger great.

10. *Pain*.—We have seen that acute pains in various parts of the body are almost universally present in the yellow fever, and may be regarded as characteristic of the disease. Considered collectively, they may be said to constitute a symptom of a suspicious nature, the danger keeping pace with the degree of their acuteness and persistence.⁷ The remark will be found applicable to each, considered separately.

A. Thus, acute pain in the forepart of the head, especially if it extend to the bottom of the orbits and is attended with a sensation of burning heat, has invariably been found to denote considerable severity in the disease, and to render the issue very doubtful—the chances of recovery lessening in proportion to the aggravation of the pain.⁸ The supervention of acute headache,

¹ Rochoux, pp. 294–5; Rush, iii. 68; R. Jackson, i. 184.

² Rush, iv. 34; R. Jackson, i. 183; Gillkrest, ii. 273; Dariste, p. 72, 174; Kelly, xiv. 381; Arnold, p. 21; Caldwell, p. 94; S. Jackson, p. 58; Pariset, p. 452; Bally, p. 275; H. McLean, p. 101; Seaman, p. 13; R. Jackson (Spain), p. 120; Copland, iii. 148; Harrison, ii. 321.

³ Jackson, i. 184; Rochoux, p. 294.

⁴ R. Jackson, i. 184; Currie, p. 37.

⁵ Caldwell, p. 96; Gillkrest, ii. 273; H. McLean, p. 102.

⁶ R. Jackson, i. 183.

⁷ Lining, ii. 428; Kelly, xiv. 380.

⁸ Stone, vi. 557; Pariset, p. 443; Seaman, p. 13; Rochoux, p. 299; Evans, p. 232; H. McLean, p. 99; R. Jackson (Spain), p. 120; Caillot, p. 161; Gillespie, p. 56; Kelly, xiv. 381; Harrison, ii. 381.

or its increase after the cessation of pain in some other part of the body, must be viewed as an indication of considerable danger.¹

B. Contrary to the experience of Dr. Rush, physicians most conversant with the yellow fever agree in opinion that pain in the loins, or rachialgia, must likewise be regarded, when carried beyond certain limits, as a dangerous sign. It has often been found to determine accurately the probable degree of severity of the case.² When of that intolerable acuteness which it but too frequently attains, the patient may be said to be in imminent peril,³ connected, as it sometimes is, with inflammation or neuralgia of the kidneys, which lead generally to suppression of urine,⁴ as well as with serious derangements of other glandular and secretory functions of the abdominal viscera.

C. The acute pains of the thighs, knees, and legs, which occur almost invariably, and are sometimes of extreme severity, as well as those of the upper extremities, which sometimes show themselves, furnish indications similar to the foregoing.⁵

D. A like danger may be apprehended from acute, burning pain at the epigastric region, and more particularly from that elicited by pressure on that region, especially when thereby nausea and vomiting are excited; here, again, the danger generally increases in direct ratio to the severity of the suffering experienced, and the extent of its continuance.⁶ On this subject, however, exceptions are not few; cases being found to end favourably in which the pain was acute and distressing; while, on the contrary, the disease proves occasionally mortal in cases in which there was no pain.

E. Pricking and shooting pain in the bowels, when very severe, is of bad omen.⁷

F. Pain in the urethra, so as to oblige the patient to pull the penis involuntarily, with or without delirium, is a fatal sign.⁸

G. Increased sensibility of the skin, especially when severe, is a very unfavourable sign.⁹

H. In other cases, an absence of pain, or a sudden cessation of it, has been found extremely dangerous.¹⁰

I. Pain in the fauces, throat, and along the œsophagus, has been noted as a sign of dangerous import.¹¹

¹ Deveze, p. 30; Bally, p. 282.

² Ralph, ii. 76.

³ Dariste, p. 173; McArthur, p. 349; Gillkrest, p. 273; Pariset, p. 443; Evans, p. 232; Stone, vi. 557; Seaman, p. 13; Caillot, p. 161; Rochoux, p. 299; Ralph, ii. 76; Jolivet, p. 12; Wragg, x. 72.

⁴ Rochoux, p. 299.

⁵ Palloni, p. 8; Stone, vi. 557; Bally, p. 282; Rochoux, p. 299; Harrison, ii. 381; Jolivet, p. 12.

⁶ Currie, p. 36; Harrison, ii. 135, 322; Kelly, xiv. 380; Pariset, p. 443; Bally, pp. 279, 280; Rochoux, p. 286; Dariste, p. 176; Fellowes, p. 61; Gillespie, p. 58; Evans, p. 232; R. Jackson (Spain), p. 121; McArthur, p. 349; Joubert, p. 972.

⁷ Fellowes, p. 61; Rochoux, p. 288; McArthur, p. 349.

⁸ Fellowes, p. 66.

⁹ Pariset, p. 443; Harrison, ii. 322; Stone, vi. 557.

¹⁰ Rush, iv. 34, 49.

¹¹ Copland, iii. 148.

κ. On the other hand, a favourable issue may be anticipated when these pains are not very severe at the outset or during the course of the disease, and when the other symptoms are not of a nature to cause alarm; as also when they disappear completely, but not suddenly, towards the second or third day, due regard being paid also to the absence of other unfavourable signs. An attention to this circumstance is of absolute necessity, as, in cases attended with severe cerebral complication, pain is not felt at the epigastrium or elsewhere, and is not complained of, though the danger is not the less urgent.¹

11. *Delirium*, in its several varieties, not being an indispensable attendant on pure and uncomplicated cases of yellow fever—patients often going through to death without exhibiting any derangement of the intellectual faculties—we may conclude that, as a general rule, the absence of such a derangement cannot, by itself, be viewed in the light of a prognostic sign of great utility, and that implicit reliance must not be placed on it unless it is taken in connection with other symptoms. But, while such is the case as regards the absence of delirium, a somewhat different inference must be drawn from its occurrence; for, as it presents itself in a large number of cases, at one period or another of the disease; as more die with than without derangement of the mind; and as but comparatively few recover who have suffered from it to any considerable extent, we may justly regard delirium as an unfavourable symptom, and its absence, on the whole, as one of a contrary character. Indeed, experience has everywhere taught the propriety of hailing with satisfaction the unimpairedness of the mental faculties, and of regarding the occurrence of delirium with suspicion, and, indeed, as a symptom of more or less danger.² At the same time it would be wrong to view it as inevitably fatal, for many recover who have been delirious during even several days. Much will depend, in respect to the indication to be drawn from the derangement of the mind, on the period at which it manifests itself, on the length of time it continues, and on the forms it assumes. Delirium may be viewed as particularly unfavourable when it occurs at a very early period of the disease.³ It has also been found dangerous when it comes on at the close of the febrile stage; and, when it breaks out at an advanced period, it is almost always the forerunner of death.⁴ It is to be borne in mind, however, that in individuals who, from peculiarity of constitution, are predisposed to, and suffer from delirium, from a slight accession of febrile excitement—and the existence of such is well known to every practitioner—the occurrence of that symptom at an early period is not a source of as much alarm as in others differently circumstanced.

¹ Rochoux, p. 290; Finlay, p. 17; Harrison, ii. 381.

² Rush, iv. 34; Arejula, Edinb. Journ., i. 450; A. Smith, p. 32; J. Clark, p. 11; H. McLean, p. 102; Fellowes, pp. 64, 65; Shecut, p. 121; Arnold, p. 21; Caldwell, p. 96; Currie, p. 36; Valentin, p. 174; McArthur, p. 349; Pariset, p. 443; Bally, pp. 275, 276; Rochoux, p. 314.

³ Lining, ii. 431; Jackson (Spain), p. 120; Currie, p. 36; Valentin, p. 174; Rochoux, p. 500; Dariste, p. 171.

⁴ J. Clark, p. 11; Harrison, p. 321.

The danger to be apprehended is greatly enhanced when delirium, having appeared at an early stage of the disease, continues during the state of remission;¹ when it is of a continuous character, and does not abate at some period of the day; or when it is of long continuance, and manifests no disposition to subside—other symptoms being at the same time unfavourable.²

Violent and maniacal delirium, whether accompanied with severe excitement in the circulation; or with irregular and feeble reaction; or marked, in the later stages, by loud laughter, singing, or other kinds of joyous expressions, is a sign of fearful omen.³ So, also, is that form in which the mind is depressed and desponding, the ideas are dull, the imagination is annihilated, the memory greatly impaired, or the patient is harassed by the idea of death;⁴ in which the mind exhibits a false idea of the state of the disease, and a consequent desire to ride out or go home (in persons who are absent from their families);⁵ in which the patient exhibits a feigned gayety;⁶ or, again, in which the delirium is of the low, muttering kind.⁷

When, on the other hand, delirium does not manifest itself, other things being equally favourable, greater hopes may be entertained. So, also, when the delirium observes a remittent character, or when it is mild and of that sort in which the ideas are lively, the imagination brilliant, and the mind inexplicably enlightened.⁸ While a cessation of delirium at any stage, especially if attended with a gradual disappearance of other unfavourable symptoms, must always be hailed as one of the harbingers of recovery. Dr. Jackson also remarks that “mild delirium, after convulsion or a state of stupor, is, for the most part, indicative of recovery.”⁹

12. *Coma* or *stupor*, whether it occurs early or at a late period of the disease, is always, especially when profound and long continued, indicative of great danger. When it supervenes in the last stage, it generally denotes a speedy dissolution.¹⁰

In the last stage of the disease, an uncommon serenity of mind, accompanied with an unusually placid countenance, will be found to be a very bad sign.¹¹ Dr. S. Jackson, of this city, remarks (at page 58): “The expression of sullen indifference, and the apparent calm and quietude that so frequently reigned exteriorly, displayed the real nature of the malady, and vividly portended the fearful storm that was to ensue.”

13. In cases attended with *convulsions*, whether these occur in the early or in the later periods, little good can be expected; for it may be doubted

¹ Caldwell, p. 96; Currie, p. 36.

² Rochoux, pp. 314, 500; R. Jackson, p. 155; Currie, p. 39; Lining, ii. 431.

³ Rochoux, p. 500; Jackson, i. 176; Currie, p. 36; McArthur, p. 349.

⁴ Jackson, i. 178–180; Copland, iii. 148.

⁵ Rush, iv. 49.

⁶ Gillkrest, p. 273.

⁷ Currie, p. 36.

⁸ Jackson, i. 178.

⁹ See Lind, p. 284.

¹⁰ Currie, p. 39; Rush, iv. 4; Cyclopaedia, ii. 273; Rochoux, p. 500; Lining, ii. 431; Jackson (Spain), p. 120; Arnold, p. 22; Caldwell, p. 95; Caillot, p. 164; Savarésy, p. 287; R. Jackson, i. 178; Dariste, p. 171; Thomas, pp. 46, 47; Harrison, ii. 135.

¹¹ Imray, Edinb. Journ., liii. 84; Arnold, p. 22; Rush, iv. 34.

whether, when they are violent and general, recoveries under those circumstances, ever occur. When they set in, in the last stage of the disease, death, in the very largest number of cases, is sure to be near at hand.¹ In some seasons, they would seem to be more certainly fatal than in others. Dr. Rush saw three recoveries after convulsions in the year 1798. All died who were convulsed in 1793 and 1797 (iv. 49).

Partial convulsions, or spasm of the legs or arms, or other parts, though less frequently fatal than general convulsions, constitute, nevertheless, symptoms of very serious omen. Spasmodic twitches in various parts, and about the mouth particularly, are indicative of imminent danger.²

With some exceptions, subsultus tendinum is in this, as in other diseases, indicative of imminent danger, unless in some individuals who, from peculiarities of idiosyncrasy or habits of living, are affected with that symptom, even in mild attacks of the disease.³

14. The same may be said of *tetanic rigidity* of the muscles,⁴ and difficulty or inability of deglutition.⁵ These symptoms are highly dangerous.

15. As to *singultus*, which, as we have seen, occurs frequently in this disease, and, as being allied to the aforesaid symptoms, may be noticed in this place, it must be viewed, especially when severe, long continued, or obscure, and when attended with anguish and restlessness, and, late in the attack, with discharges from the stomach, as a just source of great apprehension, and as generally, though not always, fatal. Under no circumstances is it to be regarded as unworthy of the greatest attention.⁶ At the same time, it must not be forgotten that in some cases recovery takes place, notwithstanding the occurrence of this symptom.⁷

Sir James Fellowes, though regarding hiccup as portending a fatal issue, remarks that this was particularly so, when the pulse fell and was low, when the extremities were cold, and the patient was light-headed. Under these circumstances, the danger—during the epidemic of Cadiz—was imminent. When, on the contrary, the pulse beat strong and the lower extremities were warm, recovery might be expected. The observation is correct.

To this it may be added that a feeling of inquietude, fear, and terror, is a sign of great danger.⁸ So also impatience and irritability of mind.⁹ Equal

¹ R. Jackson (Spain), 120; Gros, p. 17; Rush, iv. 49; Dariste, p. 179; Bally, p. 285; Fellowes, p. 66; Shecut, p. 121; Ralph, ii. 77; Caillot, p. 165; Osgood, p. 13.

² Rochoux, p. 326; Gillkrest, ii. 273; Bruce, p. 278; Townsend, p. 257; Copland, iii. p. 147.

³ Rochoux, p. 313; Arnold, p. 22; Thomas, p. 47.

⁴ Cyclopaedia, ii. 273.

⁵ Gros, p. 17; S. Jackson, p. 52.

⁶ Rush, iv. 49; Currie, p. 39; Musgrave, ix. 119; Palloni, p. 8; Pariset, p. 449; J. Clark, p. 18; Dariste, p. 179; Valentin, p. 174; Louis, p. 245; Harrison, ii. 136; A. Smith, p. 32; Shecut, p. 121; Fellowes, p. 64; Jackson (Spain), p. 121, &c.; Gillkrest, ii. 273; Ticknor, iii. 228; Lind, p. 284; Ralph, ii. 77; Arnold, p. 22; Thomas, p. 46; Caillot, p. 165; Gilbert, p. 79; Copland, iii. p. 148.

⁷ Ralph, p. 77; Fellowes, p. 64; Harrison, p. 156.

⁸ Pariset, p. 441; Shecut, p. 121; Seaman, p. 12; Deveze, p. 31; Bone, p. 28; Caillot, p. 165; Jolivet, p. 12.

⁹ Pariset, p. 442.

apprehension may be entertained when the patient lies on his side enveloped in his bedclothes, in possession of his faculties, but completely indifferent to passing events, and to the issue of his disease, and annoyed at being disturbed.¹

16. A diminution or loss of the power of vision has invariably been viewed as a highly dangerous and, indeed, fatal sign;² and, contrary to the experience of Dr. H. McLean, who did not class a deafness among unfavourable symptoms, diminished activity, or loss of the sense of hearing, has been generally held in the same light as that of vision.³

17. *Agitation, Restlessness, Anxiety, Jactitation.*—These symptoms—impatience of pressure without specification of actual pain; a constant desire to change place and posture without assignable cause; of getting out of bed and going out of doors—are threatening, and when obstinate and continuous, may be ranked amongst the signs of great danger.⁴

“A desire to ride out, or to go home, in persons who were absent from their families, was, in every instance where it took place, a fatal symptom.”⁵ “It existed to such a degree in some of the patients in the City Hospital, that they often left their beds, and dressed themselves in order to go home. All these patients died, and some of them in the act of putting on their clothes.”⁵

The subsidence of these symptoms, and their cessation at the period of metaptosis, when attended with favourable signs, may be hailed as indications of returning health. When not so attended, the occurrence is not so favourable; the calm and tranquillity observed being, under those circumstances, rather the effect of prostration and inability to move than of an amendment of the disease.⁶

18. *Sleep.*—Disturbed and unrefreshing sleep, and perhaps more particularly pervigilium, which, as we have seen, is a source of great distress in this disease, are calculated to create strong suspicions of danger. If carried very far, the latter is imminent.⁷

When, on the contrary, sleep refreshes; and, when the patient gets a few hours of it not broken in upon by vomiting; when, after being interrupted or destroyed, it returns towards the period of relaxation or metaptosis, on

¹ Gillkrest, ii. 273; S. Jackson, p. 58; H. McLean, p. 98.

² Gros, p. 17; Shecut, 121; Arnold, p. 22; Blane, p. 442; Bally, p. 276; Rush, iv. 34; Osgood, 13.

³ Davidge, p. 104; Shecut, p. 121; Arnold, p. 22; Arejula, Edin. Journ. i. 450.

⁴ Rush, iv. 34, 49; Caldwell, p. 94; Currie, p. 36; S. Jackson, p. 57; A. Smith, p. 32; Lining, ii. 431; Stone, vi. 557; Gillkrest, ii. 273; Pariset, p. 441; Palloni, p. 8; Lempriere, ii. 90; Dariste, p. 172; Rochoux, p. 301; Gillespie, p. 57; Shecut, p. 121; R. Jackson, i. 171; Copland, iii. 147, 148; Osgood, p. 13; Savarésy, p. 270; Pugnet, p. 357; Harrison, ii. 132; Arnold, p. 21.

⁵ Rush, iv. 49.

⁶ Ibid., p. 301.

⁷ Currie, pp. 36, 37; R. Jackson, i. 177; Dariste, p. 172; Rochoux, p. 305; Rouppe, p. 413.

the fourth or fifth day, hopes may be entertained,¹ provided, however, other signs of amendment occur; for the partial return of sleep at this period may be the effect of diminished sensibility and not of a real amendment.²

Heavy drowsiness, unless constitutional, is of bad omen when it occurs early in the attack. A sleepiness on the first and second days of the fever,³ and a strong desire to sleep, without the power of doing so, prognosticate an unfavourable issue. Somnolency, or stupor, which usually takes place later in the disease than the former, is always a sign of danger; and is so in proportion to the tardiness of its appearance.⁴

19. *Vertigo* and *syncope* from slight exertion are unpromising and dangerous symptoms.⁵ Little good is to be looked for when the patient lies across in bed, changes his natural manner of lying, assumes any whimsical and unusual position;⁶ and is, withal, insensible to modesty and delicacy in uncovering parts usually covered, though answering questions consistently.⁷ Dr. Rush saw but one such recover (iv. 49). When the patient lies obstinately on his back, and still more when he remains on his stomach, danger is to be apprehended. A return to a side position, other symptoms being favourable, is good.⁸

20. *Pulse*.—Of all the means of information we possess for the purpose of forming an estimate of the issue of a case of yellow fever, the pulse is, perhaps, the least to be trusted—patients not unfrequently being found in whom the arterial system is little affected even from the commencement of the attack; while at a certain period of severe and fatal cases, the pulse so frequently presents the characteristics of health as to deceive the inexperienced and impart to him the idea of approaching recovery even at a time when death is near at hand. This was early observed by Lining, who, after pointing out some of the indications derived from the pulse, remarks that there was less dependence to be placed upon it than is common in other diseases; “for, in some patients, in the second stage of the disease, even within a few hours of their death, the pulse, with respect to its fulness, softness, equality, and frequency, has continued like that of one in perfect health; although, from the other symptoms, the death of the patient could be foretold with great certainty” (p. 430). Savarésy has, in like manner, dwelt on the fallaciousness of the indications derived from the pulse (p. 289); Nassy (p. 24), Lind (p. 283)—and others might easily be added to the list—have done the same, more or less pointedly.

Nevertheless, though the pulse may often fail to impart to us the requisite information, though we are obliged to rely on other symptoms in our search for correct prognostical indications, it does not follow that in all cases, and

¹ Rochoux, p. 305; Savarésy, p. 286; Gillkrest, ii. 273; H. McLean, p. 102; Copland, iii. p. 148; Arnold, p. 17.

² Rochoux, p. 305..

³ Rush, iv. 33; Harrison, ii. 138, 321.

⁴ Rochoux, p. 306; R, Jackson, i. 177.

⁵ Gillespie, p. 56; Pariset, p. 443; H. McLean, p. 99; Rochoux, pp. 305, 503.

⁶ Fellowes, p. 66; H. McLean, p. 98.

⁷ Fellowes, p. 66.

⁸ Rouppe, p. 411; Savarésy, p. 274; Rochoux, p. 302.

under all circumstances, its condition affords no useful knowledge in that respect. So far from this, while the preservation of, or return to, its natural rhythm, does not alone always afford a clue to the issue of the case, its various deviations, in point of strength, frequency, volume, and regularity, as well as the manner in, and period at which changes in these respects take place, very generally afford grounds upon which to form an opinion, as to the severity and result of the disease.

An unfavourable prognosis may, in general, be formed when the pulse is weak, shattered, or natural at the outset of the attack—other symptoms also being of a suspicious character.¹ A like opinion is justified when at any other time it is remarkably depressed, small, languid, or vermiform;² or when it is irregular and intermittent—unless a crisis takes place, of which such a pulse is sometimes the forerunner and indication. In the last stage, that kind of pulse is always highly dangerous.³

Equal danger is to be apprehended when the pulse is very hard and full;⁴ or small, tense, hard, contracted, and vibrating;⁵ or full, but very compressible—the artery feeling as if filled with air, and yielding to the slightest pressure;⁶ or, when it is very frequent at an early—or, as sometimes, but not always, occurs—at an advanced period.⁷

Evans remarks, that a pulse of 110 in the minute is indicative of great danger; and Rochoux, after stating the same of a pulse at 120, adds that the indication furnished by its frequency is so precise, that on ten prognostics founded on that sign alone, nine will be confirmed by the issue.

Nor is the danger to be apprehended less imminent, when the pulse, whatever may have been its condition in the early stage, becomes reduced, in point of frequency, much below the natural standard—to 60, 50, or even lower—which, as we have seen, is the natural tendency in most severe or fatal cases; and indicates a sinking of the powers of life.⁸

The change, in point of frequency, is the more to be dreaded not only when it is considerable, but also when it comes on suddenly, and progresses rapidly, as is often the case at the period of the metaptosis—unless, as sometimes occurs, none of the other symptoms are indicative of danger, or the change is accompanied with a critical discharge, particularly by the skin.⁹

¹ Rush, iv. 34; Dariste, 172; Rochoux, p. 491; Caldwell, p. 95; Desportes, i. 198; H. McLean, p. 98.

² Gillkrest, ii. 273; Gillespie, p. 56; Thomas, p. 47; Ralph, ii. 76; Pariset, p. 445; Bally, p. 281; Dickinson, p. 354; Copland, iii. 148; Currie, p. 36; A. Smith, p. 32; Rochoux, p. 491; Lining, ii. 429; Fellowes, p. 64; Kelly, xiv. 380; Harrison, ii. 321.

³ Gillkrest, p. 273; R. Jackson, i. 164; Rochoux, p. 491; Gros, p. 16; Lining, ii. 427; Rush, iv. 34; Currie, p. 36.

⁴ Dariste, p. 172; Currie, p. 36; Palloni, p. 85; Rochoux, p. 318.

⁵ Rochoux, p. 318; Fellowes, p. 64.

⁶ S. Jackson, 57; Bally, p. 281; Gros, p. 16; Copland, iii. 148.

⁷ R. Jackson, i. 164; Evans, p. 243; Rochoux, pp. 318, 319.

⁸ Rush, iv. 34; Evans, p. 243; Rochoux, p. 491; Gros, p. 16; Stone, p. 555; Copland, iii. 148; Osgood, p. 13; Dalmas, p. 11; Thomas, p. 47. ●

⁹ Gillespie, p. 56; Pariset, p. 445; J. Jackson (Spain), p. 121; Rochoux, p. 491; Ralph, ii. 76.

When the pulse becomes open, free, and expanded, an unfavourable prognostic is justified, unless the change is accompanied by a critical perspiration, for, in the absence of such a discharge, it soon declines in force and expansion (*R. Jackson*, i. 162). A suspension of the pulse, for a considerable time, is an almost sure indication of death. In the exceptional cases observed by Dr. R. Jackson, the respiration was calm and free, and the eye and countenance serene (i. 164).

On the other hand, a favourable opinion of the result may be formed when the pulse—all other things being equal—is full, free, open, equal, and active in the first stage.¹ When this condition continues beyond the third day, when the pulse becomes slower, softer, and fuller from the fourth to the sixth day; when, whatever may have been its condition before, it becomes soft, expanded, buoyant and elastic near a day of crisis;² when its frequency does not exceed 80° or 90° in the minute in the opening stage;³ when it does not give way very considerably or rapidly on or about the third day, both in point of force and frequency;⁴ again, when it presents none of the unfavourable conditions above noticed; or finally, when it is neither very slow nor very rapid, nor very soft, during the second day, hopes may be entertained as to a favourable issue.⁵

The phenomena revealed by the auscultation of the heart afford prognostical signs of a highly useful character. We have seen, in speaking of the symptoms of the disease, that in an early stage the heart is often found to beat violently, but nevertheless communicates very little impulse to the ribs; that the sounds of its action are either very much subdued or one or both are entirely lost, and that ultimately a species of rumbling, or churning sound, accompanies the disappearance of the natural sounds. These signs, combined with great distress or fatigue in the breathing, and the indescribable anxiety felt at the præcordia, or under the sternum, are supposed to indicate the formation of a fibrinous clot in the cavity of the heart, and almost invariably point to a fatal termination. Dr. Pennell informs us he has diagnosed the existence of these clots several days before death in nearly a hundred cases, all of which, as he predicted, proved fatal. In upwards of fifty of these he verified, by dissection, the absolute presence of the clots. He admits that the evidence is not conclusive, but that its value is considerably enhanced by the fact that he never diagnosed the presence of a clot in a case that recovered; so that, be the explanation what it may, an examination of the condition of the heart affords a clearness and certainty of prognosis in a most insidious disease which we did not possess before.⁶

21. *Hæmorrhages*.—As a general rule, hæmorrhages may justly be regarded as a symptom of serious and even very dangerous import, especially when

¹ Caldwell, p. 96; Rochoux, pp. 317, 318; Fellowes, p. 64; Harrison, p. 321; Ralph, p. 77.

² Jackson (Spain), p. 120; Lind, p. 284; Ralph, ii. 71.

³ Rochoux, p. 318.

⁴ Gillkrest, ii. 273.

⁵ Copland, iii. 148; Arnold, p. 16.

⁶ Med.-Chir. Tr., xxxvi. 249.

they are very copious and frequently repeated. They have so been regarded by writers on the yellow fever in tropical and temperate regions from the days of Warren to our own, by not a few of whom they are held as indicative of an almost certainly fatal termination.¹ But notwithstanding the unfavourable opinion thus expressed in reference to this subject, it would be improper to consider hemorrhages as invariably and inevitably fatal. In this city and country—and experience has proved that the same observation is applicable to other yellow fever regions—cases frequently occur in which bleeding from the various outlets does not lead to fatal consequences, even when it takes place at an advanced period, and when it is of the passive kind.²

As already remarked in a former chapter, there are not wanting those who have no dread of hemorrhages in the disease before us, maintaining that they furnish no correct indications as to the probable issue of the case; and that so far from always aggravating the disease, they often prove salutary. Reference was there made to the opinions and statements of an experienced physician of the West Indies, Dr. Catel, who regards hemorrhages, at whatever period they may appear, from whatever part they may proceed, and however profuse they may be, in the light of critical discharges, provided the blood flows externally. A writer of our country, Dr. Fenner, though admitting that danger may accrue from an excess in the loss of blood, regards hemorrhage much in the same light as Dr. Catel. Speaking of the fever of 1853, at New Orleans, he says: "Hemorrhage, however, was not always an unfavourable symptom; on the contrary, it often indicated the approach of a *salutary crisis*. Indeed, it is the *natural crisis of this type of fever when its course is not interrupted by art*. How often have I hailed with joy the appearance of a moderate hemorrhage from some *safe part*, as the nose, gums, or uterus, in the critical stage of the yellow fever, when it has been previously altogether uncertain how the case was going to terminate! At this stage of the disease, a small quantity of blood flowing spontaneously from any part except the *stomach*, is most generally followed by the happiest effects, reducing any remaining febrile excitement, and allaying nervous inquietude. When hemorrhage sets in, however, there is no telling how far it may go before it stops. Believing, as I do, that it is a *natural critical discharge in this type of fever*, I would recommend that no efforts be made to check it whilst it is *within the bounds of safety*; but when it seems to threaten life, we should then resort to astringents, styptics, stimulants, and tonics" (p. 50).

¹ Dariste, p. 178; Savarésy, p. 287; A. Smith, p. 32; J. Clark, p. 11; Gillespie, p. 41; Chisholm, i. 166; Lining, ii. 431; Arnold, p. 23; Rochoux, pp. 494-5; Caldwell, p. 93.

² Blin, p. 8; Nassy, p. 26; Louis, pp. 255, 258; Pariset, p. 376; S. Jackson, p. 60; Ralph, ii. 77; Desperrière, p. 126; Rush, iii. 149; Barnwell, pp. 386-7; Deveze, p. 31; Gilbert, p. 79; Currie, p. 35; Girardin, p. 34; Gros, pp. 16, 17; Barton, pp. 46-7; Moseley, p. 449; Gillkrest, ii. 274; Seaman, pp. 13, 14; Harrison, ii. 136; Dickson, Eclect. J., iv. 110; Ticknor, iii. 228; Bally, p. 276; Caldwell, p. 93; Rochoux, p. 331; Fellowes, p. 203; Desportes, i. 198; Levacher, p. 72; Dariste, p. 177; Dickinson, p. 141.

Much, in that respect, will be found to depend on the period at which hemorrhages appear, the parts whence they proceed, the quantity and quality of the blood discharged, and the symptoms by which they are accompanied.

A. Hemorrhages which occur at the close of the first stage of the disease are often innocuous, sometimes beneficial, by removing pain and congestion of the head. They have even been stated to exercise a critical agency. Dr. Rush remarks, in relation to the epidemic of 1793, "that not a single death occurred from natural hemorrhages in the first stage of the disease" (iii. 149); and in the writings, already cited, of Gros, Fellowes, Levacher, Dariste, Dickinson, Desperrière, Gilbert, Moseley, Harrison, Bally, Rochoux, Deveze, Currie, Desportes, Joubert, Copland, we could find sufficient grounds to justify us in forming a favourable prognostic from a flow of blood at the period in question—other circumstances not being of a character to counterbalance the effect of the discharge. In mild and protracted cases, epistaxis, when it occurs on the fifth or seventh day, has often proved critical and salutary.¹ It may be remarked, however, that in some epidemics the results were different. At Dominica, in 1793, a hemorrhage at the close of the febrile stage was regarded as a bad sign.² The same may be inferred from the remarks of Arnold (p. 23); and Chisholm, as already seen, speaks of hemorrhages as being at all periods prejudicial (i. 166).

In the latter stages, hemorrhages are invariably found to indicate great and imminent danger; and indeed, except in a few instances, the harbingers of a fatal termination:³ recoveries, if they occur, doing so but seldom, and only under very particular circumstances.

B. When the flow of blood is very profuse, and the hemorrhages are frequently repeated, or when the blood oozes drop by drop—from certain orifices particularly—the prognosis is less favourable. In the former case, they endanger the life of the patient by the prostration thereby produced, and indicate a hemorrhagic tendency soon to be displayed by the appearance of black vomit; in the latter case, they indicate an adynamic or atonic condition of the system, from which recovery seldom takes place.⁴

C. Hemorrhages are the more to be dreaded when the blood issuing from the vessels is dark, black, thin, ichorous, and otherwise altered in the way mentioned in a former chapter, and especially when the parts whence it proceeds have a raw, sphacelated appearance.⁵ When, on the contrary, the fluid preserves its healthful character, or is not considerably altered, a less unfavourable prognosis may be hazarded.

D. Hemorrhages, whether in the early or at a late period of the disease, are

¹ S. Jackson, p. 60; Deveze, p. 31; Gillespie, p. 56.

² J. Clark, p. 11.

³ Caillot, p. 164; Gillespie, p. 41; Seaman, p. 13; Moseley, p. 449; Shecut, p. 121; Dariste, p. 178; Arnold, p. 23; Bally, p. 276; Rochoux, pp. 333, 494; Dickinson, p. 141; Fellowes, pp. 62, 203; Velasquez, p. 15; Deveze, p. 31; Currie, p. 39; Lind, p. 285; Gilbert, p. 78; Thomas, p. 46; Lining, iii. 431; Copland, ii. 147.

⁴ Chisholm, p. 166; Dariste, p. 178; Savarésy, p. 287; Rochoux, p. 494; Lining, ii. 431.

⁵ Copland, iii. 147.

indicative of more danger when accompanied by symptoms of an unfavourable character, or when they are not attended or followed by an amendment in the condition of the patient. In the latter stage, their association with the ordinary signs of prostration—slow and weak pulse, cold skin, hiccup, delirium, and especially with the black vomit—must be looked upon as a certain indication of a fatal issue; while in or about the early stage the prognosis is not so generally unfavourable, especially when the strength is not too much wasted or the irritation very considerable, and when the effusion of the blood occasions an amelioration to the existing condition of the patient.

E. The preceding remarks apply principally to epistaxis, the most frequent of all hemorrhages in the disease before us. Not so frequently attendant on fatal cases is that from the mouth. Unless the prostration is very considerable, and the other symptoms are of an unfavourable character, it does not constitute a source of great uneasiness, even when it appears—which it generally does—at an advanced period of the disease. At this period, indeed, it has been found (on the fifth or sixth day) to constitute a good sign, and even to prove critical, especially when the blood does not ooze at the same time from other parts. On this subject I have already dwelt (in the chapter on Hemorrhage), and need say no more about it in this place.

When, on the contrary, it is accompanied with evident signs of debility—small and weak pulse, cold skin, &c.—it is indicative of great danger.¹ The menstrual hemorrhage is favourable when the other symptoms abate.² Real uterine hemorrhage in the last stage is generally fatal, especially when profuse.³ The same results attend, except in a few instances, on hemorrhage from the anus, unless it arises from hemorrhoids.⁴

Hemorrhage from the bladder has been found occasionally to be critical;⁵ in general, however, it is a symptom of very dangerous import.⁶ Discharges of blood from the stomach and bowels, though not always fatal,⁷ are usually to be looked upon with extreme suspicion. Even a tinge of blood in the fluid ejected from the stomach at the commencement of, or early in, the attack, is dangerous; but hæmatemesis succeeding black vomit must be viewed otherwise.⁸

F. As regards internal hemorrhages, it need scarcely be remarked that petechiæ, or the small flea-bite spots which are usually considered as such, are generally indicative of considerable danger, whatever be the period at which they may appear.⁹ In some epidemics, and under peculiar circumstances, they have appeared indicative of much less danger.¹⁰ By Dr. Bally, attention has been called to the fact that when they assume a rose hue, and disappear

¹ Rochoux, pp. 333, 495; Fellowes, p. 62; Pariset, p. 445; Barnwell, p. 386.

² Lining, ii. 431; Deveze, p. 31.

³ Dariste, p. 179; Deveze, p. 31; Rochoux, p. 494; Pariset, pp. 425, 446; Audouard, p. 69.

⁴ Dariste, p. 179; Rochoux, p. 494; Bally, p. 280; Desportes, i. 167.

⁵ Desportes, i. 198.

⁶ Rochoux, p. 336, Dict. des Sci. Med., xv. 363.

⁷ Bally, p. 280; Rush, iii. 149.

⁹ Gros, p. 17; Rochoux, p. 337; Gillespie, p. 57.

⁸ Blair, p. 85.

¹⁰ Townsend, p. 153.

gradually, they are favourable; while they must be viewed in a different light when they are of a violet colour, and especially when black.¹ Ecchymoses, or stripes, or patches of a livid, green, violet, or dark colour, whether small or large, and especially when irregular in form and size, are highly dangerous, and usually, if not always, portend a fatal issue.² An equally unfavourable prognosis is to be drawn from extensive hemorrhage under the skin, in the cellular membrane, or in the substance of the muscles.³

An unfavourable prognosis must be drawn when the blood becomes sizzly and cupped, as these conditions are indicative of an inflammatory complication, which enhances the natural danger of the disease. Dr. Rochoux, who has paid much attention to this subject, says, in reference to the fever of the West Indies, that some individuals in whom the blood presents the thin, jelly-like, grayish, and tremulous size, doubtless recover, but that those in whom the fluid is covered with a dense and thick coat usually perish. Hence, it is a favourable sign to see the blood deprived of inflammatory crust, or to find the latter lessen in thickness and disappear under the use of the lancet, after having been more or less thick during the first days of the disease. When, on the contrary, we find the crust become thicker and denser at each bleeding, and assume withal a yellow hue, death is, as it were, inevitable.⁴

Still more to be dreaded is the dark, uncoagulated, and dissolved condition of the blood, which occurs in the advanced periods of the disease. It is usually, if not always, associated with symptoms of prostration, and always of dangerous import. This want of cohesion and dark colour of the blood is, of course, an unfavourable sign when it occurs, as it does sometimes, early. When the dark colour of the fluid disappears, and the latter brightens under the use of the lancet, the indication is favourable.

Free effusion or oozing of blood from wounds, blistered surfaces, leech-bites, &c., is a dangerous sign.

22. *Stomach*.—The degree of danger in the yellow fever, may, in general, be estimated by the extent and obstinacy of nausea and vomiting, which, as we have seen, are the almost inevitable attendants, from first to last, of the disease.

A. When occurring to a great extent in the early stage; when they manifest a tendency to increase and bring no relief, those symptoms indicate considerable danger, and are usually the forerunners of the fatal changes. Continued nausea is a symptom of more suspicious import than full and free vomiting, unless other phenomena are favourable.⁵

¹ Typhus d'Amérique, pp. 284, 285; Gros, p. 17.

² Jackson (Spain), 121; Lining, ii. 431; Evans, p. 239; Rochoux, pp. 338, 498; Fellowes, p. 63; Cyclop., ii. 273; Gros, 17.

³ Pariset, p. 447; Rochoux, p. 339; Gillkrest, ii. 273; Catel, p. 227.

⁴ Fièvre Jaune, pp. 159–161; F. Amaril, p. 342.

⁵ Jackson, i. 170; H. McLean, p. 99; A. Smith, p. 32; Dariste, p. 172; Palloni, p. 8; Seaman, p. 13; Arnold, p. 20; Lining, ii. 429; Kelly, xiv. 380; Currie, p. 36; Caillot, p. 162; Gillkrest, ii. 273; Hunter, p. 94; Blane, p. 436; Gillespie, p. 58; Pariset, p. 451; Bally, p. 278; Rochoux, p. 281; Rush, iv. 33; Dickson, p. 353; Lind, p. 283; Joubert, p. 970.

B. Their advent, increase, and continuance during the second stage—that of remission or metaptosis—or their appearance at that period, when they did not exist before, is equally, if not more, unfavourable.¹

C. Great danger may be apprehended when these symptoms are attended with an acrid and burning sensation, and a feeling of anxiety at the præcordia.²

D. The discharge of green porraceous bile at the commencement, or during the first stage of the attack, or that of drinks unchanged or mixed with ropy mucus, is indicative of considerable danger. The ejection of pure bile, however large the quantity may be, is less so. The danger increases when the matter ejected becomes of a brownish or grayish colour, or assumes a slate, chocolate, or sooty tint; especially when, at the same time, it is tinged with blood, abundant and thick, or, as we have seen, when it consists of pure blood.³

E. Equal danger, if not more, is to be apprehended from the discharge by the stomach of the white ropy acid fluid, which, as we have seen, is frequently ejected at the close of the second stage, and has received the name of premonitory or precursory fluid or white vomit. It is thrown up occasionally in considerable quantities, and after much retching, but though affording much relief to the symptom, the effect is fallacious and the discharge is soon succeeded by an aggravation of the disease.⁴

F. These changes are the forerunners of the black vomit of the closing stage, which, if not invariably indicative of approaching death, is, as already seen, sufficiently often so to justify its being regarded as a fatal sign; care being taken to ascertain that the blood thus ejected is the product of effusion from the gastric vessels, and not from those of the nostrils, gums, and pharynx, swallowed and afterwards thrown up.

G. When, on the other hand, the stomach is tranquil during the first stage, or when nausea and vomiting have not been very troublesome; when there is no vomiting during the stage of remission; when, having existed, these symptoms subside from the 3d to the 5th day, and finally disappear (without the continuance of epigastric anxiety and pain; for, in the latter case, black vomit may be expected), and when the matter thrown up consists of simple mucus, or yellow or green, or even black bile, or improves in quality, a favourable prognosis will be justifiable.⁵

G. Hæmatemesis succeeding black vomit proves often a favourable sign, the undecomposed blood probably indicating, as Dr. Blair properly suggests, a cessation of the acid formation (p. 85).

¹ Lining, ii. 430; Arnold, p. 20; Currie, p. 36; Caldwell, p. 96; Harrison, ii. 322.

² R. Jackson, i. 171; Blane, p. 438; Lempriere, ii. 90; A. Smith, p. 32; R. Jackson (Spain), p. 121; Kelly, xiv. 380; Gillkrest, ii. 273; Rochoux, p. 480; Dariste, pp. 172, 173; Osgood, p. 13.

³ Evans, p. 234; Lempriere, ii. 88; R. Jackson, i. 170; Rochoux, pp. 277, 478; A. Smith, p. 32; Blair, p. 85.

⁴ Blair, p. 80.

⁵ Rush, iv. 32; Moultrie, p. 13; Palloni, p. 8; O'Halloran, p. 84; A. Smith, p. 32; S. Jackson, p. 59; Dariste, p. 175; Rochoux, pp. 281, 479; Caldwell, p. 96; Ralph, ii. 71; Bone, p. 28; Copland, iii. 148; Lind, p. 284; Ralph, ii. 77.

II. The supervention of a sensation of hunger in the last stage, other unfavourable symptoms being present, is a fatal sign.¹

The occurrence of eructation or explosions of wind from the stomach indicates a state of considerable danger.²

I. An extension of the sensation of rawness and burning along the course of the œsophagus to the fauces, is equally to be dreaded.³

23. *Bowels*.—A. Obstinate constipation, and insensibility to the action of purgatives and enemata, at the onset of the disease, are, and have at all times and everywhere been accounted unfavourable signs.⁴

B. When, as sometimes happens, diarrhœa sets in during the first stage, danger may be apprehended if the stools are green, porraceous, brown, dirty-looking, fetid, or thin. Equal danger is incurred when stools of that character are obtained by artificial means.⁵

C. Fears may be entertained, also, when evacuations give no relief in the second stage;⁶ or when diarrhœa sets in towards the close.⁷

D. An unfavourable prognosis is justified when the stools are black and tarry, viscous and small in quantity;⁸ or whitish or clay-coloured;⁹ or muddy, like turbid coffee or charcoal and water, with shreds of mucus; or like dissolved liver; as well as when the matter is like black, grumous, and dissolved blood, with or without coffee-grounds, or like pure blood, or when from black it becomes red; or when the stools are fetid, and attended with meteorization.¹⁰

The coffee-ground stools above noticed, or involuntary passages and tenesmus, are highly dangerous, the first two generally fatal, especially when connected with typhoid symptoms.¹¹

Finally, the suppression of diarrhœa on the third or fourth day is a sign of bad omen; the result being generally the supervention of coma and other symptoms of equal danger.¹²

On the other hand, when the bowels are freely and easily opened;¹³ when

¹ Rush, iv. 34, 49; A. Smith, p. 32; S. Jackson, p. 57; McArthur, p. 349; Blair, p. 89.

² R. Jackson (Spain), p. 121; A. Smith, p. 32; Gillespie, p. 58; Pariset, p. 451; Bally, p. 278; Rochoux, p. 282.

³ Kelly, xiv. 380; Gillkrest, ii. 274.

⁴ Rush, iv. 33; Seaman, p. 13; R. Jackson, i. 172; Dariste, p. 173; Currie, p. 36; Blane, p. 440; Bally, p. 280; Desportes, i. 199; Jackson (Spain), 120; Chisholm, i. 173.

⁵ Gillespie, p. 58; Lining, ii. 431; Rochoux, p. 284; Deveze, p. 32; R. Jackson, p. 173.

⁶ Dariste, p. 284.

⁷ Rush, iv. 33.

⁸ Jackson, p. 173; Spain, p. 120; Lining, ii. 431; Desportes, i. 199; Blane, p. 440; Dalmas, p. 76; Caillot, p. 162; Bally, p. 280; Osgood, p. 13; Joubert, p. 970; Savarésy, p. 287; Currie, p. 39; Rouppe, p. 409; Dariste, p. 76.

⁹ Rush, iv. 33; Currie, p. 36; Gillkrest, p. 273; Blane, p. 440; Kelly, xiv. 381; Osgood, p. 15.

¹⁰ Bally, p. 280; R. Jackson (Spain), p. 120; Dariste, p. 176; Gillespie, p. 58; McLean, p. 79; Kelly, xiv. 381; Rochoux, p. 284; Dariste, p. 173.

¹¹ Gillespie, p. 58; Kelly, xiv. 381; Arnold, p. 23.

¹² Deveze, p. 32; Caillot, p. 162; Savarésy, p. 287.

¹³ Deveze, p. 32; Ralph, ii. 77.

the stools are loose and bilious, or dark and excremental, or naturally discharged without being highly offensive to the smell, or when they are of natural consistence and colour, the prognosis must be favourable;¹ as, also, when from black the stools become brown;² when there is a free discharge of bile, or feculent matter, toward the fifth, sixth, or seventh day, or at the period of metaptosis;³ or when a diarrhœa sets in before jaundice has occurred, or towards the close of the stage of irritation; such evacuations often prove critical.⁴

24. *Tongue*.—The appearance of the tongue does not always afford a correct indication of the probable issue of the disease. It is, indeed, one of those fallacious guides to which allusion has been made, and is regarded as such by every one conversant with the yellow fever. Many individuals, even in cases of long duration, go through the disease to death or recovery without exhibiting any, or if any, very inconsiderable alteration in the tongue, which remains moist, and, at times, scarcely more red on the edges or furred in the centre, than we find in health, or in indispositions of the most trifling character.⁵ The absence, therefore, of those changes which usually indicate danger is not of very great value in the present disease, unless when combined with other favourable symptoms.⁶ It does not the less follow, however, that the alterations in the appearance of that organ described in a former chapter furnish, when they do occur, useful prognostical indications.

A. An unfavourable prognostic may safely be hazarded when the tongue is soft, flabby, swollen, covered with a glutinous saliva.⁷

B. When it is clean, smooth, red, glossy, or shining, whether dry or moist.⁸

C. When it is pale and thin, covered with a thin coating throughout, or in patches, of white fur—broad, slightly red on the edges; the red shining through the white fur.⁹

D. When it is red on the edges and at the tip, with a tendency to become dry in the centre.¹⁰

E. When it is clammy, after the stage of reaction has subsided.¹¹

F. When it presents a thick yellow, orange, or brown coat or stripe of

¹ Rochoux, p. 285; Palloni, pp. 8, 9; Stone, vi. 563; H. McLean, p. 102.

² Moultrie, p. 16.

³ Gilbert, p. 78; Currie, p. 35; Bally, p. 280; Osgood, p. 15; Copland, iii. 143.

⁴ Moultrie, p. 16; Dickinson, p. 141; Currie, pp. 23, 35; (Fev. of 1797), p. 219; Pugnet, p. 360; Rochoux, pp. 479, 480; Deveze, p. 46; Rush, iv. 32, 33; Fontana, p. 73; Gros, p. 17; Cathrall, p. 36; Maher, p. 893; Desperrières, p. 71; Berthe, p. 123; Palloni, p. 8; Dalmas, p. 233; Monges, ii. 58; Merrill, ix. 247; Lind, p. 285; O'Halloran, p. 130; Audouard, p. 59; Blin, p. 11; Desportes, i. 196, 197; Jackson, i. 160, 173; Blane, pp. 412, 414; Osgood, pp. 14, 15; Lempriere, ii. 89; Gillespie, p. 40; Moseley, pp. 448, 449, 468, 469; Towne, p. 22; Gilbert, pp. 73, 78; H. McLean, p. 95; Caillot, p. 158; Bally, p. 473; Pugnet, p. 356.

⁵ Rush, iv. 41; S. Jackson, p. 52.

⁶ Rochoux, pp. 327, 477.

⁷ Dariste, p. 174; Jackson (Spain), p. 120.

⁸ R. Jackson, i. 168; Kelly, xiv. 380; Rochoux, p. 327; Copland, iii. 148.

⁹ Jackson, p. 120; Evans, p. 244; Pariset, p. 451.

¹⁰ Evans, p. 244; Pariset, p. 451.

¹¹ Harrison, ii. 322.

the same kind in the commencement of the attack, and when, withal, it is dry.¹

G. When it assumes a leaden hue, while its edges are red, and it is divided longitudinally in the centre,² or the colour which remains after drinking red wine, with little movement in it.³

H. When it is rough and somewhat foul—the foulness adhering to the surface; or when it is covered with a black crust, or sanguineous exudation, or puckered and red on the edges—the danger increasing with the rapidity of the change; or when the organ presents a dark streak in the centre, with white edges.⁴

I. When it is dry, shrivelled, rough as a file, or red, as if roasted, with a brown, dark, or black fur in the centre.⁵

K. When it is tremulous, affected with spasmodic movements, difficult to protrude; and when the patient forgets to draw it in again.⁶

L. When it is, or becomes, moist and natural, while symptoms of a dangerous or malignant kind present themselves, or are in no way amended, the chances are decidedly against the patient. Generally, indeed, the change is of fatal import.⁷

A favourable opinion of the issue may be entertained if the tongue retains its natural appearance and moisture, or is but slightly red or coated; if it is gray or white, without the supervention of any untoward symptoms; if the changes it may undergo are effected slowly and gradually; or if, having been affected in some of the ways mentioned, the organ cleans from the edges, loses its redness, and reacquires its normal volume and moisture.⁸

To this I may add that a preternatural secretion and excretion of mucus from the glands of the throat, discharged, as Dr. Rush remarks, by an almost constant hawking and spitting, is of favourable omen.⁹

25. *Thirst*.—The absence of thirst, when combined with symptoms of a suspicious or dangerous character, affords no reasons for a favourable prognosis, as cases frequently occur in which the disease progresses to a fatal termination without exhibiting any change from the standard of health in regard to the desire for drink. When associated, as it is sometimes, with a dry and rough tongue, or with high febrile excitement, the total absence or the existence of a very limited degree of thirst, indicates a state of con-

¹ Rush, iv. 49; Fellowes, p. 61; Kelly, xiv. 380; Caldwell, p. 74.

² Frost, xiii. 13; H. McLean, p. 99; Rush, iv. 34; Fellowes, p. 63.

³ Fellowes, p. 63.

⁴ Jackson, i. 166, 167; Kelly, p. 380; Deveze, p. 29; Arnold, p. 20; Rochoux, p. 476; S. Jackson, p. 52.

⁵ Jackson (Spain), p. 120; *Ib.* Sketch, i. 166; Arnold, pp. 17, 20; Gillespie, p. 58; Pariset, p. 450; Bally, p. 278; Gros, p. 16; Stone, vi. 558; Arnold, p. 17.

⁶ Jackson, i. 168; Pariset, p. 450; Townsend, p. 149; Gros, p. 16; McLean, p. 99.

⁷ Rush, iv. 49.

⁸ Bally, p. 277; Arejula, *Edinb. J.*, i. 450; Arnold, p. 17; Rochoux, pp. 325, 477; H. McLean, p. 102; Rush, iv. 33; Harrison, ii. 321; Copland, iii. 148.

⁹ Rush, iii. 63, and iv. 33; Osgood, p. 14; Ralph, ii. 79.

siderable danger.¹ An equally, if not a more unfavourable prognosis, may be drawn when thirst is excessive, especially when the tongue is at the same time natural, or when this symptom occurs at a period at which it ought to have abated;² and when there is a constant desire for drink, with a smacking of the lips—the patient taking little at a time.³

But, however dangerous these symptoms may prove, they are not fatal when the black vomit has not occurred.

On the other hand, a moderate degree of thirst, or a diminution of it when other signs are not of an alarming character, and more particularly when these are decidedly good, affords room for a favourable prognosis.⁴

26. *Respiration*.—In the early period of an attack of yellow fever, and, indeed, throughout the progress of the case, slow respiration, interrupted and intermixed with deep heavings, is justly regarded as highly dangerous. The same opinion may be formed when, while the febrile action runs high, there is a state of calm, and, as it were, still respiration, with scarcely perceptible expansions and contractions of the chest.⁵ Difficulty of breathing—whether the function be accelerated, or the reverse—and a sense of weight and oppression about the chest, are of dangerous import, and have ever been so considered.⁶

The most suspicious of all the conditions of respiration, as Dr. R. Jackson has well remarked, abstracted from conditions that belong to direct disorganization of the lungs, consists in a sense of stricture—a desire to expand the chest without the power to effect it, and without restraint in doing it from a sense of local pain or impediment; more especially when accompanied by a livid or very deep crimson of the countenance, or by a dull, torpid, and lurid aspect (i. 165).

When the difficulty of breathing is associated, as is, indeed, usually the case, with deep sighing or moaning, the danger is rendered more apparent.⁷

Tremulousness, shrillness, and feebleness or extinction of the voice, as well as slowness of articulation in the latter stage, are very bad, and, indeed, fatal signs;⁸ and a loud monotonous wailing has properly been accounted equally so.⁹

A dry, hoarse, and sore throat, was followed by death in every case in which it occurred in Dr. Rush's practice during the epidemic of 1798 (iv.

¹ R. Jackson, i. 168; Rochoux, p. 323.

² A. Smith, p. 32; R. Jackson, i. 168; Rochoux, p. 324; Currie, p. 36.

³ R. Jackson, i. 168.

⁴ H. McLean, p. 102; H. Smith, p. 32; Rochoux, p. 324.

⁵ Jackson, i. 165.

⁶ Arnold, p. 21; Fellowes, p. 66; Caldwell, p. 94; Currie, p. 37; Dariste, p. 172; Blane, p. 439; Pariset, p. 449; Bally, p. 281; Palloni, p. 85; Osgood, p. 13; Bone, p. 28.

⁷ Currie, p. 37; Caldwell, p. 95; Kelly, xiv. 380; R. Jackson, i. 165; Dariste, p. 172; Arnold, p. 21; Gillkrest, p. 273; Blane, p. 439; Pariset, p. 449; Rochoux, p. 291; Harrison, ii. 132, 322.

⁸ Savarésy, p. 273; Caillot, p. 165; Copland, iii. 148; Dariste, p. 179; Currie, p. 37; Rochoux, p. 327.

⁹ Gillkrest, p. 273; Rochoux, p. 292.

49). According to Dr. Townsend, the latter symptom was invariably fatal at New York in 1822 (p. 160).

In 1793, the same symptom was regarded in a very different light by Dr. Rush, and was one of the circumstances which led him to use a salivation in that fever (iv. 49).

27. *Odour of the Breath*.—We have seen that the breath in the yellow fever has often a peculiar, disagreeable, alliaceous odour. This may serve as a useful prognostic sign.

“Many years ago,” Dr. Stevens informs us, “I remarked that a man, named Benson, who had long kept a hospital, in the island of St. Thomas, for the reception of sick sailors, was, in this disease, almost invariably right in his prognostics. In some cases of this fever, where the patients might have appeared to a common observer to be in little danger, for in these there was no local pain in any of the solids, nor any increase of heat, or increase of action in the vascular organs, yet he prognosticated, from the first, that these patients would not live. In other cases, where they were suffering severely from violent excitement, he as pointedly declared that if these patients were well bled and properly treated, there would be no danger. In one case, when I found him very positive where I thought him wrong, I asked him by what criterion he judged that he was so certain about the result. He said, that in that fever, he only required to smell the breath to know whether the patient was to live or die. That in cases which were really bad, although they might not appear to be so, there was always a peculiar putrid smell in the breath, and that when this existed to a certain degree, he had never once known them to recover.” Dr. S. adds that he since found this to be an excellent, though very disagreeable test, but that he has known patients recover who had this peculiar odour to a great degree.¹

28. *Skin*.—The indications furnished by the skin must be considered under several heads: 1, those derived from the temperature; 2, from the functions; 3, from the diseased conditions of that membrane.

A. *Temperature*.—A favourable prognosis may be hazarded when, in the early stage, the skin is animated, and the temperature is moderately above the natural standard, and devoid of pungency; as also when it is equably diffused over the body, limbs, and extremities, and continues so during the first forty-eight hours, or beyond the usual period of remission, whether or not from the effect of treatment or other influential agencies.²

On the other hand, when the heat is concentrated, deep seated, pungent, or acrid, and in excess, or of the character denominated *ardor mordicans*; when it retains that character during several days, and when it is irregularly distributed over the surface, or confined to the head, neck, and breast, the prognosis is highly unfavourable, the danger increasing in pro-

¹ On the Blood, pp. 222–3.

² Jackson, i. 184; H. McLean, p. 102; Arejula, i. 450; Cyclop., ii. 277; Ralph, ii. 76, 7; Copland, iii. 148.

portion to the elevation and pungency of the heat, which cannot much exceed 105° F. without being followed by a fatal issue.¹

Equally or more unfavourable is the prognosis when the surface, in the early stage, retains its natural warmth, or is cooler than natural, and imparts no glow of warmth to the hand, the patient, at the same time, complaining of internal heat; and when, at the metaptosis, it becomes suddenly cool, without free perspiration, and afterwards gradually attains a temperature below that of the atmosphere, and even becomes preternaturally cold.²

B. *Functions of the Skin*.—Danger may be apprehended when there are dryness, harshness or roughness of the skin, combined with considerable heat;³ when likewise the skin is damp, clammy, greasy, and torpid or relaxed; and the perspiration, though copious, is viscous or adhesive;⁴ when, withal, the skin is pale, and when copious perspiration breaks out at the commencement of the attack—especially if, notwithstanding the skin is hot, imparting to the hand the feeling of a hot substance sprinkled with water, and the other symptoms remain unabated.⁵ The prognosis is also unfavourable, the issue doubtful, if not usually fatal, when at any time the skin, while cool, is wet; when the sweat is “cold, clammy, and partial, or scanty, and accompanied with a frequent, small, and unenergetic pulse,” and other bad symptoms. When associated with the black vomit, these latter conditions of the skin are of fatal augury.⁶

The prognosis, on the contrary, is favourable when, in the early stage, and during the first forty-eight hours, moderate heat is combined with natural moisture and softness of the skin;⁷ when, in mild cases, a gentle perspiration, occurring early, and kept up for a day or two, is attended with an amendment of the other symptoms; when, at the period of remission, or from the fifth to the ninth day, and especially towards morning, perspiration breaks out; when it flows easily without being forced, and is copious, warm, and diffused all over, diminishing the ardor of the skin, and being accompanied with other good signs. In many such cases perspiration proves critical.⁸

C. *Diseases and other Alterations of the Skin*.—Diminished sensibility of the skin, insusceptibility to irritation; or when, vesication being attempted, the parts become dry, seared, and withered (*R. Jackson*, i. 184–5; *S. Jackson*,

¹ *R. Jackson*, i. 184–5; *Ib.*, *Fev. of Spain*, p. 87; *Gillespie*, p. 57; *Evans*, p. 244; *Arnold*, 7, 22, 23; *Rochoux*, p. 322.

² *Rush*, iv. 34; *Copland*, iii. 148; *Evans*, p. 244; *Pariset*, p. 449; *Kelly*, xiv. 380; *R. Jackson (Spain)*, p. 121, &c.; *Ralph*, ii. 76; *Bone*, p. 28; *Osgood*, p. 13.

³ *Rush*, iv. 34; *Jackson*, i. 184–5; *Dariste*, p. 172; *Stone*, vi. 558; *Jackson (Spain)*, p. 12; *Lind*, p. 283; *Bone*, p. 28.

⁴ *R. Jackson*, i. 185, 187; *Stone*, vi. 558; *Copland*, iii. 148; *Bone*, p. 28; *Dickson*, p. 354.

⁵ *Bancroft*, p. 47; *Berthe*, p. 103; *Rochoux*, p. 322.

⁶ *Caillot*, p. 164; *R. Jackson*, i. 186.

⁷ *Cycl.*, ii. 273; *Jackson*, i. 184.

⁸ *Berthe*, pp. 102–3; *Rush*, iv. 33; *Pugnet*, p. 360; *Deveze*, p. 31; *S. Jackson*, p. 60; *Osgood*, p. 15; *Gillespie*, p. 57; *Caldwell*, p. 96; *Bally*, p. 281; *Moseley*, p. 449; *Copland*, iii. 148; *Dickinson*, p. 141; *Jackson*, i. 186; *Finlay*, p. 17; *Fellowes*, p. 53, *note*; *Ralph*, ii. 71; *Arnold*, p. 17.

pp. 52-4); increased sensibility (*Stone*, p. 557); a relaxed, flaccid, and inelastic condition of the skin, are symptoms of the gravest import—the first is fatal (*R. Jackson*, i. 185; *Ib.*, *Fev. of Spain*, p. 120; *Kelly*, xiv. 330). A rash, or eruption of a miliary character upon the skin;¹ boils, or small abscesses;² parotids, attended with a healthy suppuration;³ an eruption occurring on the fifth and seventh day upon the lips and about the mouth, rising freely, assuming the suppurative process, and producing pain, are favourable signs.⁴ When, on the contrary, these eruptions do not rise freely, or when they turn hard and dry (*R. Jackson*, i. 187); when the body emits the cadaveric, or peculiar and disagreeable odour mentioned already;⁵ when anthrax, carbuncles, petechiæ, vibices, and dark livid patches, aphthæ, and local gangrenes of the scrotum, penis, &c., make their appearance, or when blistered surfaces become livid, or cease to suppurate, the prognosis is unfavourable.⁶

29. *Urine*.—A. When, in the early stage of the disease, the urine is high-coloured, hot, burning, and scanty, the indication is inauspicious; its great scantiness being often the precursor of suppression.⁷

B. Black, or very dark and offensive urine is indicative of considerable danger, but does not uniformly portend a fatal issue.⁸ The same may also be said of chestnut-coloured, and especially of bloody urine, with or without clots;⁹ of an involuntary discharge of the fluid, and of a sediment appearing on the first or second day—the danger being in proportion to the quantity deposited.¹⁰

C. Suppression of urine from a cessation of secretion has everywhere been recognized as a fatal sign, few, if any recovering in whom the symptom presents itself.¹¹

¹ *Cycl.*, ii. 273; *Fellowes*, p. 60.

² *Warren*, p. 17; *Desportes*, i. 198; *Gilbert*, p. 78.

³ *Desportes*, i. 198; *Osgood*, p. 15; *Townsend*, p. 153; *Blanc*, p. 439; *Copland*, iii. 148; *Catel*, *An. Mar.*, p. 227.

⁴ *H. McLean*, p. 102; *R. Jackson*, i. 187; *Blanc*, p. 439; *Roupe*, p. 412; *Moseley*, p. 449; *Arnold*, p. 19; *Imray*, liii. 83; *Gilbert*, p. 78.

⁵ *Gillespie*, p. 59; *Gros*, p. 17; *Ralph*, ii. 75; *Caillot*, p. 164; *Copland*, iii. 148; *R. Jackson*, i. 187.

⁶ *Fellowes*, pp. 56, 63; *Deveze*, p. 52; *Rochoux*, p. 498; *Townsend*, p. 153; *Copland*, iii. 147-8; *Joubert*, p. 971; *Harrison*, ii. 135; *Lind*, p. 283; *Savarésy*, p. 289.

⁷ *Dariste*, pp. 173-6; *Rochoux*, pp. 509-10; *Jackson*, i. 173; *Blanc*, p. 440; *Pariset*, p. 452; *Dariste*, p. 173.

⁸ *Rush*, iv. 34; *Osgood*, p. 13; *Jackson*, i. 174; *Blanc*, p. 440; *Copland*, iii. 148; *Dariste*, p. 173; *Pariset*, p. 441.

⁹ *Rush*, iv. 34; *Lining*, ii. 431; *Pariset*, p. 451; *Jackson (Spain)*, p. 120; *Arnold*, p. 23.

¹⁰ *Lining*, ii. 429.

¹¹ *Arejula*, p. 187; *Drysdale*, i. 136; *Rochoux*, pp. 310, 510; *Fellowes*, p. 63; *R. Jackson (Spain)*, p. 120; *Rufz*, p. 16; *Dalmas*, pp. 9, 11; *Desportes*, i. 199; *A. Smith*, p. 32; *Lining*, ii. 431; *J. Clark*, p. 18; *Dubreuil*, viii. 323; *Gros*, p. 16; *Palloni*, p. 8; *Dariste*, pp. 135, 176; *Caldwell*, pp. 82, 93; *Valentin*, p. 174; *N. O.* 1839, p. 325; *Cartwright*, ix. 11, 33; *Caillot*, p. 164; *Savarésy*, p. 287; *Harrison*, ii. 135, 321; *Maher*, p. 880; *Monges*, ii. 58; *Pariset*, p. 451; *Bally*, p. 281; *Rush*, iii. 62, iv. 34; *Pugnet*, p. 360; *Gilbert*, p. 79; *Baxter*, xxi. 3; *S. Jackson*, p. 56; *Ralph*, ii. 74; *Osgood*, p. 13; *R. Jackson*, *Sketch*, i. 174; *Velasquez*, p. 15; *Dickson*, p. 354; *Copland*, iii. 148; *Jolivet*, p. 12; *Thomas*, p. 46.

D. Finally, when the urine appears like serum, with small particles apparently of lymph floating through it, the whole being very coagulable, without either urinous or aromatic odour, and without urea, death will ensue; this condition of the fluid being as fatal as its suppression.¹

E. On the other hand, a favourable opinion of the issue of the disease may often, though not invariably, be formed, when, whatever be its colour, the urine is secreted and voided plentifully, naturally, and without pain in the urethra.²

F. Equally favourable is the prognosis when the urine has no highly offensive odour;³ when it is thick, turbid, and deposits a sediment at the moment of metaptosis, or when late in the attack, it is sometimes critical.⁴ Hopes may also be entertained when the urine becomes of a deep yellow colour at those periods in which jaundice is favourable.⁵

G. Strangury has also been found, at times, to be a good sign,⁶ while a bloody discharge from the bladder has sometimes proved critical, especially when it is copious, and ceases soon and completely.⁷

H. To this it may be added, that though scantiness and coagulability of the urine are highly dangerous signs, the patient may escape, provided the fluid retain its urinous character.⁸

30. *Strength*.—A sudden and considerable loss of strength at the commencement of the attack, or at the period of remission, is an unfavourable sign—the danger increasing in proportion to the extent of the prostration.⁹ The same indication is furnished when, in the course of the after stages, the prostration is considerable, and especially when it is accompanied with tremors of the limbs when moved, and a tendency to faint on slight exertion.¹⁰ When, however, the other symptoms, at the period of remission, wear a very favourable aspect, muscular debility need not be viewed as a cause of alarm,¹¹ though, in general, the absence of prostration at that period is of favourable omen.¹² At the same time, it does not follow from this that a retention of muscular strength must necessarily be a good sign, as many patients retain a large share of it up to the moment of death, or regain it during the latter periods of the fatal attack, when prostration had occurred before. In the latter cases, the exhibition of muscular strength, amid other symptoms of dangerous import, constitutes a highly unfavourable sign.

¹ Pennell, *Med.-Chir. Trans.*, xxxvi. 252.

² Kelly, xiv. 380; Rochoux, pp. 331, 512; Blane, p. 440; Copland, iii. 148; Finlay, p. 17; Gillkrest, ii. 273; H. McLean, p. 102.

³ McLean, p. 103.

⁴ Desportes, i. 196; Jackson, i. 174; Gillespie, pp. 57–8; Pugnet, p. 380; Currie, p. 37; Osgood, p. 15; Townsend, p. 155.

⁵ Towne, pp. 62–3; Rochoux, p. 509; Stone, p. 562.

⁶ Kelly, xiv. 380; Rush, iii. 207; Dickson, p. 354.

⁷ Jackson (Spain), p. 123.

⁸ Pennell, *loc. cit.*, p. 252.

⁹ H. McLean, p. 99; Palloni, p. 85; Kelly, p. 380; Lining, p. 429; J. Clark, p. 18; Osgood, p. 13.

¹⁰ H. McLean, p. 99; Rochoux, pp. 305, 503; Gilbert, p. 79.

¹¹ Rochoux, p. 303.

¹² J. Clark, p. 18.

When the patient lies on his side, and draws the bedclothes around him, the prognosis is favourable; when, on the contrary, he lies on his back, and keeps uncovered, or when he lies on the edge of the bed, with his head drooping over, danger may be surely apprehended.¹

CHAPTER XXIV.

INCUBATION.

WHATEVER may be the nature of the cause giving rise to the yellow fever, some difference exists as to the length of the period of its incubation. By this is to be understood the time which elapses from the moment of the effective action of the cause to that of the manifestation of the disease—that period during which the system tolerates the poison, and the latter remains apparently innocuous until reaction is brought about by some intrinsic circumstance of the depressing kind, or otherwise. The period of incubation, as thus defined, differs from that which elapses from the first moment of exposure to the morbid agent to the appearance of the disease; for in many individuals, who, from various circumstances, are for the time, or permanently, protected from the disease, such exposure is innocuous, and the cause is eliminated from the system without leaving any traces behind. Nor must we confound it with what Hildenbrand and others have called the opportunity of the disease—*i. e.* the period characterized by the phenomena portending to, or premonitory of, the outbreak of the attack. The latter period is one of diseased process; that of incubation is one of latency, during which there is no appreciable manifestation of a morbid condition, but which is sure to be followed, sooner or later, by the open development of the fever. It may be said to terminate at the accession of the other—the opportunity—which constitutes part and parcel of the disease.

That, after the effective impression of the cause, and prior to the manifestation of the morbid product of that impression, as exhibited in the outbreak of the disease, whether the attack be of a serious or slight character, some abnormal modification takes place in the solids or fluid, or both, is a point which we are bound to believe. In ordinary cases, in which the introduction of the cause proves harmless—in which the individual, though exposed to its action, remains unaffected—we must suppose that the morbid agent, though floating in the system, produces none of the changes alluded to, and is as constantly thrown off or eliminated as introduced; the organs being, from absence of predisposition, the result of peculiarity of conformation, or of modification produced by acclimatization or prior attacks, protected from its morbid effects. But in those in which, through the agency of an exciting cause, or that of the

¹ Harrison, ii. 321; Copland, iii. 148.

intrinsic force of the poison, the introduction of the latter is followed, after a period of longer or shorter duration, by a decided attack of the disease; or by the manifestation of phenomena of a premonitory kind, leading, sooner or later, to the production of the more genuine symptoms; or, in some instances, subsiding from the effect of treatment or the curative effects of nature—the idea of perfect innocuousness, during the period of latency, is scarcely admissible. Were it otherwise, we would find it difficult to explain how the morbid agent could remain so long—days, and even weeks—without being, like other foreign bodies, eliminated from the system; or how, after circulating harmlessly, for a greater or less time, in cases no longer exposed to its influence, it could all at once be aroused from its slumbers and produce a decided effect. That in some instances the result may be attributed to the sudden influence of a strong exciting cause, or a change in the constitution fitted to impart force and activity to the poison, and to arouse the susceptibility of the organs, is doubtless true; but, in many cases, the disease breaks out generally at night, some or many days after exposure, without any appreciable exciting influence, or even some time after the application of one or more of such influences, and without any apparent change in the constitution capable of allowing the poison greater scope of action. In others, the exciting cause is applied too long after exposure to allow us to believe that the poison all the while was harmlessly floating in the system. In such instances, therefore, we must admit that, during the period of incubation, some changes are produced in the system, which, though inappreciable to the senses, and putting no obstacles to the healthful performance of the functions, are not the less real; and that these changes increase, quickly or gradually, till at last the disease finally explodes. But, as regards the nature of those changes, and as to what takes place in the system during the period of latency of the poison, we know, and can know nothing, and it would be useless to inquire.

In endeavouring to ascertain the duration of the period of incubation in the yellow fever, we encounter difficulties of no inconsiderable kind. When, for example, we turn for information to contagionists, we discover that, though they are decided in their statements on the subject, we can derive but little assistance from them in the solution of the problem; for they generally take into account only exposure to the effluvia from the sick, whereas the same individual may have, and probably has, been exposed to the morbid influence of the poison for some time before approaching a patient labouring under the disease, and it is to that exposure, and not to the inhalation of the effluvia issuing from the sick, that his attack is to be attributed. Again, the period of incubation is with difficulty ascertained among those who have been for some length of time in an infected district, and continue to reside there up to the moment of their attack; for in these we cannot ascertain the precise time at which the poison, which they have imbibed in their system from the outset of their exposure, has taken effect. Nay, the same difficulty presents itself in many cases in which the individual, after being exposed for some time to the infection, leaves the place, and sickens elsewhere; because he may have received the morbid impression several days before leaving, and in calculating

from the period of his departure we would arrive at an erroneous conclusion. All that we can ascertain with certainty in such cases is, that the incubation has not been *shorter* than the period intervening between the departure of the individual from the place of infection and the manifestation of the opening symptoms. The only instances in which we can hope to arrive at a correct conclusion in the matter are those in which an attack takes place after a brief exposure to the tainted atmosphere, and thereby enables us to date with precision the period of infection. It may be remarked here that contagionists, admitting the truth of the statement regarding the regularity of the period of incubation in contagious diseases, and its irregularity in common malarial fevers, refuse, for reasons easily understood, to recognize the propriety of placing the yellow fever in the same category with the latter, which they properly regard as devoid of contagious properties, and insist on including it among those possessing those properties, on the ground that in it the incubative process, so far from being of uncertain and undefined duration, is as regular in these respects as it is in the whole tribe of contagious diseases.

On this subject, however, facts lend no support to the theory; for, under whatever circumstances the disease may appear, the period of incubation in the yellow fever varies somewhat in different cases. In this respect, indeed, the disease allies itself to those arising from malarial effluvia, while it differs in no small degree from such as are contagious, in the greater number, if not all, of which the period of latency is generally well marked and defined. Thus, Sir W. Pym states, in regard to the epidemic of Gibraltar in 1804, that "the fourth day was generally the time the contagion seemed to require to show itself," and that "this was ascertained most positively in innumerable instances" (p. 24). But it will be easy to show, on the testimony of contagionists themselves, that on this point, as indeed on most others connected with the disease, Sir William has greatly erred. At the same time, it would perhaps be hazardous to found an argument in favour of the non-contagious character of yellow fever on the irregularity as regards the limit of time within which it shows itself after infection, and on the difference of results observed in that respect in diseases possessing contagious properties, inasmuch as experience shows that the period, though more fixed in the latter than in malarial non-contagious fevers, is not so precisely thus as not to admit of considerable latitude. Reasons may also be found for believing that though in the yellow fever the length of the incubative process varies more than it generally does in some truly contagious diseases, it does not do so more evidently than in others that non-contagionists of the purest water regard as communicable from the sick to the well; and that, whatever time may elapse, in yellow and other malarial fevers, between the period of exposure and the manifestation of the symptoms, the incubative process is governed by much the same laws as govern the period of latency of all other morbid poisons. To this subject, attention will be called as we proceed.

Whatever be the place where the yellow fever shows itself, instances of short incubation are frequently noted. Ferguson says of Monk's Hill: "It was the duty of the white troops, in both forts, to take the guard and

duties of the dock-yards among the marshes below : and so pestiferous was their atmosphere, that it often occurred to a well-seasoned soldier, mounting the night-guard in perfect health, to be seized with furious delirium while standing sentry, and when carried back to his barracks on Monk's Hill, to expire in all the horrors of the black vomit, within less than thirty-six hours of the first attack."¹

Strangers who arrived at Gibraltar during the epidemic of 1804, were, in several instances, attacked on the second and third day after their landing.² Mr. Amiel says, indeed, that people were seized on the first day of their landing;³ and from this and the fact that persons who fled from the garrison were taken ill on the coast of Portugal many days after, he is induced to believe that the disease may be excited into action at any time from the first moment of exposure to a period of fifteen or twenty days. Arejula regarded the incubative process as varying from twenty-four to thirty-six hours, sometimes extending to two or three days, in a few to six or seven, and even as late as thirty or fifty days.⁴ According to Drs. Pariset, Bally, and François, the period of incubation at Barcelona, in 1821, appeared very short. "We have," they say, "very strong reasons to suspect, and those reasons are founded on facts, that this period does not exceed twenty-four hours, or three days, at most. More commonly, indeed, it is terminated in six or eight hours" (p. 487). Dr. Mazet, who was a member of the commission sent to Barcelona, arrived there on the 9th of October, and sickened on the 12th. Dr. Pariset remarks, in reference to this case, that the incubation could not have been of longer duration than twenty-four hours; because Dr. Mazet had not seen patients before the 11th (p. 488). This, however, is begging the question of the contagiousness of the disease. Thinking, as we must do, that his approaching the sick had no influence on the event, we perceive that the incubation could not have been longer than three days, as he was not exposed to the infected air of the city before the 9th. On the same occasion, some of the individuals sent to the Hospital of Observation, were taken sick on the first day, some on the seventh or eighth, and others were attacked as late as the fifteenth day.⁵ Dr. Riseuno, in a letter to Dr. Burnett, relative to the yellow fever observed by him at Carthage, in 1804, 1810, 1811, and 1813 remarks, that in those who exposed themselves to the action of the poison, and afterwards withdrew to some distance, the disease, if they were infected, made its appearance within the space of twenty days exactly. At the expiration of this period, they were perfectly safe.⁶ Dr. John Hunter, long ago, called attention to the variableness of the time which elapsed in the West Indian yellow fever, between exposure and sickness. The men on the watering service, he remarks, were not all taken ill at the same time: "Some fall sick the first or second day, and others not till several days after they have ceased to be exposed to the cause of fever, by returning on board of ship" (p. 152).

¹ Marsh Poison, in Vol. of Recollections, pp. 194, 5.

² Pym, p. 24; Bancroft, p. 249.

⁴ Pariset (Fever of 1819), p. 10.

⁶ Burnett, p. 240.

³ Johnson, p. 273.

⁵ Audouard, p. 423.

“Some have embarked on board of ship in good health, and have been seized after ten or fourteen days,” and “examples in this way have come to my knowledge, of the fever appearing three weeks after ceasing to be exposed to the cause of it” (p. 153).

In speaking of fevers from bad air, among which he includes yellow fever, Lind says: “From comparing many instances of people who have slept on shore during the sickly season—and, in consequence of it, who alone have been taken ill out of a whole ship’s company, then lying in an open road—it appears that some are immediately seized with the sickness or delirium; many are not seized with them till they have been on board for two or three days; several have been only slightly indisposed for the first five or six days; and, in a few, the symptoms of indisposition have not appeared before the tenth or twelfth day” (pp. 201, 202). “I have known,” he says, “the whole of a boat’s crew seized next morning after sleeping near the mangrove bushes, which are frequently found along the sides of the rivers of the torrid zone” (pp. 78, 81). Dr. Wallace, on the other hand, is of opinion that, generally speaking, the disease does not show itself until at least ten or twelve days after the morbid impression has been received; that oftener it is a fortnight or three weeks; and that even a later period is not uncommon (p. 273). Dr. R. Jackson states, that a detachment of Dutch artillery arrived at Port au Prince in August. The men had all been well during the passage from England. On the night after landing, eight men were attacked, seven of whom with fever (*Outlines*, p. 77). In other cases, he thinks the disease attacks much later—on the seventh or fourteenth days, or even six weeks or two months after *exposure* (p. 247).

M. Moreau de Jonnès cites a case which would seem to indicate the possibility of a very prolonged latency. The patient, a sailor, embarked on the 4th of March, 1809, at Fort Royal, Martinique, and left St. Pierre on the 14th. During the interval, he was not ashore. He sickened at sea on the 1st of April. If we suppose he carried the seeds of the fever from the former place, the incubative process was of twenty-eight days—if from the latter, of seventeen days. But, as the disease does not appear to have existed, except sporadically, at St. Pierre, and as the individual was not on shore there, we may presume he imbibed the poison at Port Royal, where the fever was prevailing at the time” (p. 123). At Demerara, in 1837, Dr. Blair witnessed instances of individuals dying of the yellow fever within one week of their arrival in the colony—having been attacked within the first twenty-four hours after exposure to the foul air of the place. He mentions the case of a mother and two daughters who embarked on board of ship and were exposed to river influence only the night previous to departure; all in good health. They sickened at sea fourteen days after (p. 69). In a case mentioned by the same writer, the individual was exposed to an infected locality on Friday, and sickened on Sunday (p. 69). In the case of a Dr. Reed, the period was the same. In another case, the period seemed extended to four months (pp. 69, 70). Bancroft states that the disease not uncommonly attacked on the fourth or fifth day after exposure; but oftener on the ninth,

twelfth, or fifteenth day (p. 238). Chisholm, on the contrary, found that whatever the predisposing cause might be, the *contagion* always acted within four days from its application to the body—in six, twenty-four, or forty-eight hours; in others, not before the expiration of the fourth day—the whole affording a medium length of two days.¹ Others have stated the period to vary from two to twelve days—possibly, sometimes longer.² M. Bertulus, who saw disease in the West Indies, limits the period to from three to eight days, and affirms that it never extends beyond three weeks.³ Dr. Stevens, also, who is well acquainted with the true yellow fever, which, in accordance with his views respecting its origin and nature, he denominates African typhus, states that it never produces its effects previous to the fourth day, and that he has known some well-marked cases in which the poison being applied in a less concentrated form, the individuals were not attacked until twelve days had elapsed after exposure.⁴ According to Dr. Finlay, the average of the stage or period of incubation is from six to ten days (p. 11).

In New York in 1822, according to Dr. Townsend, the period of incubation was, for the most part, from three to six days (p. 288). Dr. Harrison, of New Orleans, remarks, that persons who arrive in that city during an epidemic, from the healthiest region, even by the ocean, are subject to attack on the sixth, fifth, fourth, or even as early as the third day, after their arrival. Cases of attack on the third day were not uncommon during the epidemic of 1837.⁵ Dr. Merrill, lately of Natchez, and now of Memphis, in a letter with which he favoured me on the subject (Sept. 6, 1850), states that at the Bay of St. Louis, in 1820, several persons arrived when the disease was at its height, and were taken sick in twenty-four hours. "The disease was epidemic, I think, in 1827, at a plantation three miles from Natchez, when several white persons died with black vomit, and every soul on the place had the disease; and it had actually ceased to exist when a white woman of about thirty-five, enjoying fine health, arrived there from Cincinnati, slept one night, and the succeeding night was attacked by the disease, and died in forty-eight hours." Dr. Merrill states, that according to his recollection, the disease at Natchez mostly occurred from the third to the eighth day after exposure—that he has attended persons at each of these periods. He has known of none which have occurred sooner than three days; while, it is believed by some persons, that cases have occurred as late as twelve or thirteen days. He once attended an individual, who was attacked at Natchez on the fourteenth day from his departure from New Orleans, where the disease prevailed epidemically.

Similar observations have been made in this city. In his account of the epidemic of 1793, Dr. Rush says: "The seeds of the fever, when received into the body, were generally excited into action in a few days. I met with several cases in which they acted so as to produce a fever on the same day

¹ Manual, p. 198.

² Med.-Chir. Rev. and Journ., i. 395.

³ Mém. sur l'Intoxication Miasmatique, p. 39.

⁴ Stevens on the Blood, p. 235.

⁵ New Orleans Journ., March, 1847, p. 564.

in which they were received into the system; and I heard of two cases in which they excited sickness, fainting, and fever within one hour after the persons were exposed to them. I met with no instance in which there was a longer interval than sixteen days between their being received into the body and the production of the disease" (iii. 48). In 1798 it was observed that some went out of the city but one day previous to being attacked. But generally they were attacked about three, and sometimes six days after. In some instances, the disease was not taken till they had been out ten, twelve, sixteen, and even twenty-one days.¹ From all the facts I have myself been enabled to collect, I am of opinion that, though sometimes very short, and at others very long, the period of incubation in yellow fever usually varies from five to ten days.

Thus it will appear, that here, as elsewhere, the period of incubation in the yellow fever is very various; that some systems can, for a long period, throw off or resist the morbid agency, while, with others, exposure the most casual and temporary, produces immediate effect, the interval varying, as it would appear, in different epidemics, and among different individuals during the same epidemic, and extending from twenty-four hours to twenty or more days, in some to several weeks, and in a few to several months. In this respect it approximates, as already remarked, to marsh and other malarial fevers. Dr. Bryson (p. 151), remarks, in relation to the African fever, that on board of the ship *Wanderer*, the boats of which had been detached up the rivers Nunez and Pongor, with but five exceptions the attacks took place within a fortnight after the men had returned to the ship. In other instances, the attack is stated to have taken place on the twelfth, fourteenth, fifteenth, and twenty-second day (p. 152-3, 233). The yellow fever allies itself also to the ordinary remittent bilious fevers of this and other countries; to typhoid fever; to the oriental plague; and, indeed, to most zymotic diseases of a really or supposed contagious and of a non-contagious character, in all of which the incubative process is usually restricted within much the same limits observed in the former. But, though such may be the fact under ordinary circumstances, instances are not uncommon in which the period of incubation in malarial fevers particularly, has extended considerably beyond the furthest limits assigned to it in the yellow fever. Examples in point are found in the writings of Dr. John Hunter, of Bancroft, Blane, Williams, Ferrus, Paterson, Boudin, Anderson, Lee, Chapman, Drake, and others. They have been referred to in detail in another place by the author of the present work,² and will again be reverted to in a future chapter.

The late Dr. R. Jackson was of opinion that the aptitude to receive the morbid impression of the cause of fever takes place more at particular periods than at others; that it manifested itself more frequently about the fourteenth day after communication with an infected source; and that it is observed chiefly

¹ Condie and Folwell, p. 97.

² *Pneumonia: its supposed Connection, Pathological and Etiological, with Autumnal Fevers, &c.*, p. 381, &c.

at septenary periods, the seventh, fourteenth, twenty-first day, &c., from the time of exposure. This opinion he based upon the results of his own observations "made upon numerous bodies of men; upon healthy men placed as attendants in infected hospitals, and upon healthy soldiers sent to concentrated sources of endemic fevers. Among such, fever scarcely ever appeared before the seventh day, commonly not before the fourteenth, and, in numerous instances, not till the expiration of six weeks, or even two months, though the cause of disease, during this time, was ordinarily in great activity" (*Outline*, pp. 247-8). Whether this doctrine, which applies as well to remittent and intermittent as to yellow fever, which Dr. Jackson continually confounds together, will bear the test of observation, time must determine. Sufficient is it for our present purpose to call attention to the fact, that, should the statement of Dr. Jackson and others, relative to the period of attack after exposure, be well founded, it furnishes an illustration of the connection between the period of latency and the septenary revolutions of the system, so far, at least, as the number of those revolutions have been counted. To this I must add, that individuals who relapse with fevers, do so, in all probability, in consequence of the cause not being completely eliminated from their system, and remaining in a latent state during the period of exemption. If this be admitted, and we find that these relapses take place at regular septenary periods, and if we bear in mind the reasons assigned for considering the period of the recurrence or exacerbation of the symptoms, as well as that of the critical efforts of the system, as being, in like manner, regulated by the law of septenary revolution, we would appear to be justified in inferring that the process of latency—intimately allied, as it is, to the other phenomena—must also be under the control of the same law.

Dr. Bancroft is of opinion, that, *cæteris paribus*, the disease will be most violent in those cases where it appears soonest after the morbid cause has been applied to the body; and that the rapidity will be in proportion to the quantity, and concentration, and force of the noxious miasm (p. 243). The same view has been adopted by Dr. Merrill, and several other physicians of this country. By others, however, it has not been found to stand the test of experience, not only as regards the yellow, but also the concentrated fever of the African coast; long incubation not being followed always by mild attacks, and the severity of the disease not always arguing a short period of latency. By Dr. Wallace attention has also been called to the fact, that the period at which the disease shall break out, does not appear at all, or at least in any material degree, to depend upon the length of time the patient has been exposed to the poison—exposure for one night, or even less, being followed by fever as certainly, as soon, and as violently, as if the exposure had been greatly prolonged.¹ Experience here and elsewhere would seem to lead to the same conclusion.

Much may probably depend, as regards the duration of the incubative process, on the age, sex, temperament, race, and state of health of the indi-

¹ Edinb. Med. J., xlv. 273.

vidual, the period of the epidemic at which the eases occurred, &c. In the oriental plague, these eircumstances have been found to exereise an influence in that respect, and, from analogy, we may presume that the same occurs in the yellow fever ; but, so far, the question has not been aseertained by positive observation.

CHAPTER XXV.

MORTALITY OF YELLOW FEVER.

THE reader need scarcely be informed, that the yellow fever, wherever it has assumed the epidemic form, has fully established its claims to being classed among the most formidable diseases to which the human body is liable. This is true whether we view it in reference to the changes it very generally occasions in the domestic arrangements of a large portion of the exposed population ; to the great sacrifices of interest and comfort it entails on these—the necessary effects of the interruption or cessation of commercial and other pursuits ; of the abandonment of home and of the sundering of ordinary ties and occupations—to the perversion of the better feelings of our nature, to which it too often gives rise ; or to the immense loss of life it occasions, as well, proportionately, to the amount of the population at large as to the number of the sick. In this latter respect, no disease, the black plague of the fifteenth century, and the Asiatic cholera in our own days excepted, can compare with it. The oriental pestilence itself, though it occasioned, a century or two ago, a frightful mortality throughout many parts of Europe, and though it has given rise, up to this day, at each epidemic return, to a mortality equally large, in proportion to the number of individuals attacked, did not, at the time of its widest diffusion, or if it did has long since ceased to produce, as great a loss as has resulted from the disease in question. It is, and has ever been, more circumscribed in regard to the sphere of its prevalence, and has appeared less frequently, even in its more legitimate localities. While the yellow fever has, for the reasons mentioned, been viewed as an object of dread, and a fruitful source of public and individual distress wherever it has prevailed epidemically, a statement of the events noticed during the several visitations of the disease in the city of Philadelphia, will show that, besides entailing on the population at large all the ills and inconveniences to which I have adverted, it has here exercised its destructive power to an almost unprecedented extent.

It is doubtless true that the more enlightened views relative to the origin, mode of propagation, and means of prevention of the disease, entertained here at present by the members of the profession, and which have, to a certain extent, forced their way to the attention of the city authorities, and of the more influential classes of the community, have led to the adoption and

enforcement of sanitary regulations which, aided as they are by the restricted area of the infected localities, have tended latterly to render the mortality less extensive than it was in the early epidemics, and still continues to be in other places, and thereby to lessen the alarm, distress, and pecuniary loss experienced in calamities of the sort. But although the disease has by such means been shorn of a large share of its power of diffusion and rendered less formidable in an epidemical point of view, experience has but too plainly shown that it has invariably assumed, in this city, its most malignant form; that when once fairly established, it has, even under the most favourable circumstances, been the source of considerable suffering among a large portion of the population; and that while the number of cases has doubtless been rendered much smaller than it would otherwise have been, the disease has occasioned, in the later epidemics, as large a mortality, proportionately to the numbers attacked, as it did in early times.

Omitting here a consideration of the many other calamitous effects produced by the wide diffusion of the malignant fever in question, I propose to present a brief survey of the mortality to which it has given rise on various occasions, both in the aggregate of deaths recorded, and, as often as it is possible to do so, in the proportion of these to the number affected. The subject is one of no ordinary interest, as it enables us to ascertain the position which the yellow fever of this city has occupied in respect to malignancy and its power of destruction at the several periods of its prevalence, as well as the difference between it on those points, and the same fever as it has shown, and continues to show itself, in other parts of our country and in foreign lands.

To those who have turned their attention to this subject, it must be evident that, in the present state of our knowledge, such a comparison, so far as most of our epidemics are concerned, can only be approximative. In early times, no records were here kept of the number of deaths occasioned by the disease, or if they were, they have long ceased to exist, and the statements handed down to us consist, generally, of little more than the surmises, or rough estimates of contemporary writers. At a later period, a knowledge of the number of burials was obtained from the returns of the various churches; while, subsequently, a register of the deaths was kept by the sanitary authorities of the city. But at no time, if we except a few epidemic seasons, are we informed officially of the number of individuals affected with the reigning disease; owing to which we are left in the dark relative to the proportion of deaths to recoveries, or must reach that knowledge by circuitous and consequently unsatisfactory processes. It is true that in the accounts of the disastrous epidemic of 1793, and of some subsequent years, the number of admissions and deaths at the fever hospital are recorded. But cases so admitted constitute only a fractional portion of the whole number of sufferers, and the record in question, though serviceable in leading us to form some estimate of the fatal issue of the disease among the inmates of that establishment at a particular season, cannot enable us to arrive at satisfactory and correct conclusions relative to the same issue in the city at large, where

experience has shown that the mortality was sometimes greater, and at others smaller, than in the wards of the former.

Nor are we more successful in our endeavours to collect precise and accurate information on this subject in other parts of this country and in foreign regions; for while in some places and on some occasions, the requisite details are carefully noted, it more frequently happens—in situations too where a different course might naturally have been anticipated—that the information afforded is not of a nature to satisfy the scientific inquirer. Nevertheless, limited as the materials at our command often are, enough can be collected, both at home and abroad, to serve a useful purpose.

The number of deaths reported to have occurred from the fever, during the epidemic of 1699, when the population of the city did not exceed 3,800, amounted to no less, according to the writers of the period, than 220, or one in 17.3 of the entire number of residents. While the infant city, then in the 17th year of its existence, was thus sustaining a loss which, due regard being paid to the limited amount of its population, must be viewed as very considerable, the mortality in proportion to the number attacked, appears to have been unusually large. On this subject, however, we have not the means to arrive at positive conclusions, for we nowhere find mention of the number of those that were affected with the disease. Story (p. 221), in the brief notice he has left of this epidemic, states that few, if any, houses were free from the sickness; and as the number of these, including stores and public buildings, amounted to about six hundred, we may presume that the number affected did not exceed five hundred. This is the more probable, as by another writer, Monington,¹ we are told the disease was of a highly malignant and fatal character, few living *seven days after they were taken sick*, and *very few* recovering. Supposing the estimate correct, the mortality must have amounted to one in 2.3.

The loss during the more circumscribed epidemics in 1741, 1744, and 1747, is involved in too much obscurity to be used as an object of comparison. Lind, on the voucher of an anonymous letter-writer, states it to have amounted, in the former year, to 200, and, as was mentioned in an early part of this volume, Mr. Peters states it as not exceeding 250. They are, however, our only authorities on this subject; and their statements are doubtless as conjectural as, and not more authentic than, those concerning the loss occasioned by other early epidemics everywhere. But admitting them to be correct, and bearing in mind, at the same time, that the population in 1741 could not have reached above 10,000, it will follow that, on the occasion in question, the loss was about one in fifty. Of the number attacked and of the ratio of mortality among the sick, we can ascertain nothing. Nor are we better informed respecting the number of deaths and the degree of diffusion of the disease in 1744 and 1747. All we learn regarding the latter season is, that for a time upwards of 20 burials took place every day; that the disease caused much alarm, and engrossed the conversation of all companies; that

¹ Coxe's Med. Mus., i. 227.

the city early became a melancholy place; and that all who could emigrated to the country. From all this we are justified in the conclusion, that the mortality from the fever this year was considerable, and, considering the number who had fled from the city, greater, in proportion to the population, than it had been in 1741, but smaller than in 1699. All this, however, is merely conjectural, and affords no material for comparison with anterior or subsequent epidemics, here or abroad.

Not less uncertain is the estimate of the mortality from the epidemic of 1762. Mr. Thos. Willing, in a letter to Dr. Griffiths,¹ estimates the loss, in the vicinity of the Sugar-house wharf (in Southwark), at upwards of sixty; but this estimate cannot embrace the entire number of those who perished from the disease on that occasion; for the late Dr. Rush, who in 1762 was a student of medicine, mentions, in a note-book kept at the time, and from which extracts are given by him in the history of a subsequent epidemic, that the fever spread like a plague, carrying off daily, for some time, upwards of twenty persons. It should be stated, also, that Dr. Redman, a distinguished practitioner of the period, and at one time President of the College of Physicians, in a communication to this body, states that the disease commenced early in August, and did not terminate before November; that for some time he attended twenty patients daily; that other physicians had more, and that the fever was of a very malignant character. A disease which continues to reign during three entire months—which spreads like a plague—affects a sufficiently large number of individuals to enable many physicians to attend twenty or more patients a day—proves highly malignant in character—occasions, during a portion of the time of its prevalence, a daily mortality of twenty, and carries off sixty in a single locality, may well be supposed to have caused the loss of a thousand individuals. Such, indeed, is the amount mentioned in a note written by me more than twenty years ago, but the authority for which I cannot now discover. Holding this to be the probable number, and bearing in mind that the estimated population of the whole city, at that period, was somewhat less than twenty thousand, we find that the mortality from the fever must have been equal to 1 in 20 of the entire inhabitants. As regards the precise number of cases which occurred, we have no information.

More disastrous was the next epidemic, which broke out thirty-one years after (in 1793). On that memorable occasion, which forms an epoch in the history of the city, the disease carried off little less than one-twelfth of the entire number of inhabitants, the loss reported being 3,548, and the estimated population between 45 and 50,000, or 1 in 12.4; or, according to another calculation, in 6,327 houses, containing 40,144 inhabitants, 3,293 perished—a proportion of 1 to 12.5. Large as this loss must be viewed, it will be found to have been still more excessive when we deduct from the population the number who left the city from an early period of the epidemic. Amounting to more than 12,000, the emigration allowed the disease to bear

¹ Facts and Observations, by the College of Physicians, p. 10.

on an aggregate of some thirty-five thousand inhabitants, and to produce, therefore, a mortality of 1 in about 10. It is to be recollected, however, that the census of 1790 gives a total of 40,000, which, at the usual rate of increase, would raise the population in 1793 to more than 50,000. The mortality, according to this estimate, would be 1 in 12.13.

The hospital records, the only ones at all explicit on the subject, exhibit a mortality of more than one-half of the cases admitted—the number received, between the 22d of September and the 6th of November, being 807, and the deaths 448, or 1 in 1.801. Whether the proportion was equally great in the city at large, is now, and has always been, impossible to ascertain, as no mention is made, in any published document to me known, of the whole number of cases that occurred during the prevalence of the epidemic. Dr. Rush laments the large proportion of deaths to recoveries at the hospital; and, while appearing to refer this result to the kind of practice there pursued, leads us to infer that the success in the hands of other physicians, particularly of those who had adopted the therapeutical views he so eagerly recommended, was far more satisfactory. He affirms that his remedies cured a greater proportion than ninety-nine in a hundred of all who applied to him on the first day of the disease, before the 15th of September; that the same means were equally successful in the hands of other physicians, and of his pupils (ii. 165); and that, though less successful after that period, he never, then or at any other time, lost more than one in twenty of those he saw early and attended throughout. It may be doubted, however, whether genuine cases were, anywhere, at any period, and under any circumstances, during the epidemic in question, of so mild a character as to be so readily cured, and whether the result obtained was of a nature calculated to justify a glorification on the part of any physician or class of physicians. When we bear in mind that the disease, in little more than three months, destroyed the lives of, at the very least, one-thirteenth of the entire population, and of one-tenth of those exposed; and that, in a well-situated, managed, and provided hospital, it caused a mortality of more than one-half the patients admitted—we cannot suppose it to have been otherwise than exceedingly fatal, and to have carried off everywhere a large portion of the sick. Taking these circumstances into consideration, and recollecting what has been usually the course of events during other epidemic seasons, here and elsewhere, we cannot greatly err in regarding the mortality in 1793 to have amounted to no less than one-third, if it did not reach to one-half, of those attacked. Such was the result in the practice of the late Dr. Monges, who enjoyed a very large share of public confidence at the time, and was noted for his skill in this and indeed every other disease. From him I have often heard that his loss, on an average, amounted, in 1793, to one in three of those placed under his care; and if in his hands the fever could prove so fatal, it is not likely to have been found considerably less so in those of others. But, even were I not able to appeal to so good an authority, I should be disposed to arrive at the above conclusion, from the circumstance that a belief in a different and much more successful result in the practice of the majority of fully-occupied physicians would lead to the

admission that the sickness that season extended to a larger proportion of the population than it has usually been found to affect; in other words, that the cases were multiplied to an extent unprecedented in the annals of epidemics—due regard being paid to the amount of the population and to the extent of the mortality. Assuming that the latter did not exceed one-third of those attacked, and recollecting that the deaths by the fever amounted to about 3,550, we have at once a total of 10,650 cases, or nearly one-third part of the remaining, or about one-fourth of the entire number of inhabitants. It is not probable that the number could have been larger. It is true that, by Dr. Currie, we are informed that at one time there appeared to be more than eight thousand persons confined with it; and Dr. Rush tells us that not less than six thousand of the inhabitants of Philadelphia probably owe their lives to purging and bleeding during the autumn. But these are conjectural statements, and cannot be viewed as militating against the conclusion hazarded above. On the other hand, the fever runs its course in a few days; and, as it continued to prevail during three months, it must necessarily, had it really affected at the same time as many as one-fifth of the exposed population, have spared none who came within reach of its baneful influence. This we know not to have been the case. Again, if the therapeutic agents eulogized by Dr. Rush had been as extraordinarily successful as he affirms, and if they had been the means of saving the lives of six thousand individuals, either the whole mortality must have occurred in the practice of those who pursued a less energetic course—and these were the majority—or the whole population must have passed through the disease; neither of which can we admit. It cannot but strike the reader that if many physicians had been sufficiently successful, during the first month, to save ninety-nine out of one hundred of those who applied early, and at no time to have lost more than one in twenty; and that if, notwithstanding this, the mortality did not fall short of 3,500—the number of the sick must have equalled that of the inhabitants! Such being the case, the reader, if he bears in mind what precedes relative to this epidemic and to those of former years, will probably feel a reluctance in coinciding in opinion with Dr. Rush, when he remarks that, “could a comparison be made of the number of patients who died of the yellow fever in 1793, after having been plentifully bled and purged, with those who died of the same disease in the years 1699, 1741, 1747, and 1762, the proportion would be very small in the year 1793, compared with the former years; and that, including all who died under every mode of treatment, the mortality was probably less, in proportion to the population of the city and the number of persons who were affected, than it was in any of the other years mentioned.”

The fever of 1794 was of a transient character, and caused the death of only twenty or thirty individuals. For this reason, and because we have no information of the number of cases that occurred—and cannot, therefore, ascertain the ratio of deaths to recoveries—it need not occupy our attention here.

The mortality in 1797 was estimated, by Dr. Rush, at between one thousand and eleven hundred. Condie and Folwell (p. 64) state it at nine hundred and eighty-eight; Ruston (p. 64), at twelve hundred and ninety-two; while Dr.

Pascalis, who obtained his information from the records of the Board of Health, computes it at thirteen hundred. Presuming some of the deaths to have been occasioned by other diseases, we may safely estimate the loss by yellow fever at twelve hundred. The entire population amounting that year to about sixty thousand, the ratio of deaths to the entire number of inhabitants was as one to fifty, a number much less distressing than that recorded in former epidemics. It is to be borne in mind, however, that the emigration this year commenced as soon as the existence of the disease was known, and was much larger than it had been in preceding seasons—amounting, according to Dr. Rush, to two-thirds of the population. Supposing this estimate to be correct, it follows that the force of the disease bore not on sixty thousand, but on twenty thousand inhabitants, the balance having placed themselves beyond the sphere of the infection. The loss, therefore, was 1 in 16.6. But, even making our calculation on the basis of this greatly reduced population, the ratio of deaths to the number exposed was smaller than in former years.

As regards the number of persons affected with the fever, and the proportion of deaths to recoveries among them, we learn nothing. At the Fever Hospital, the loss amounted to one hundred and eighty-four; but we nowhere discover a record of the number admitted with the disease. All we can collect respecting the ratio of the mortality, is contained in a remark by Dr. Currie, in his account of the epidemic of 1799 (p. 27), from which we infer that the loss in that institution must have been at the rate of 1 in 1.60, or a little less than one-half.

The more ample and satisfactory records of the Board of Health, during the memorable epidemic of the year following, 1798, enable us to arrive at a fuller knowledge of the disastrous mortality which occurred on that occasion. From these, it will be found that the loss, as reported, amounted to three thousand six hundred and forty-five. But when to this amount we add the deaths that occurred in the country, after exposure to the infected localities, the entire mortality may be supposed to have reached four thousand. Of the number reported, five hundred and fourteen deaths took place at the Fever Hospital, and three thousand one hundred and thirty-one in the city and districts. The whole number of inhabitants amounted, at the time, to about sixty thousand, which, when divided by the reported mortality, gives 1 death in 16.48; or, if we base our calculation on the entire loss, four thousand—those who perished in the country constituting part of the population—we have 1 in 15.5. This proportion, which, under any circumstances, would justly be deemed large, sinks almost into insignificance when we take into account the extent of the emigration which took place that season, and commenced even before the official announcement of the prevailing disease. By Condie and Folwell, the chroniclers of this epidemic (p. 55), it is computed that forty thousand individuals sought refuge in the pure air of the neighbouring towns and country; leaving a comparatively small number exposed to the infection, and to share the loss occasioned by it. Supposing this estimate of the great reduction of the population to be correct, the number exposed throughout the whole duration of the epidemic did not exceed twenty-two

thousand, which, divided by the number of deaths reported, gives us a ratio of 1 to 6; a proportion exceeding that of any previous epidemical season in Philadelphia, and equalled by only a few, and surpassed by none of the epidemics of other cities of this or foreign countries. Even if we base our calculation on a smaller reduction of population, supposing all who left the city not to have done so in time to escape a thorough exposure—say twenty thousand instead of forty thousand—we have a ratio of 1 to 10.5, which, though less, is still very appalling, and has not often been exceeded in the history of the yellow fever of large cities.

While such was the heavy mortality in the city and districts, the loss in proportion to the number affected was, perhaps, still more distressing, and would alone suffice to illustrate the highly malignant character of the disease. From the 18th of August, when the first reports were made, to the close of the epidemic, four thousand seven hundred and eighteen cases were reported. Prior to that period, it is presumed that one hundred and fifty cases occurred, giving a total of four thousand eight hundred and sixty-eight. Of these, eight hundred and ninety-eight were admitted into the hospital, and three thousand nine hundred and seventy treated in the city and districts. If now we revert to what has been said of the mortality in the hospital, in the city and districts, and in all these places combined, we shall find that the proportion of the entire loss—three thousand six hundred and forty-five—to the entire number of cases—four thousand eight hundred and sixty-eight—was 1 to 1.34. The proportion in the city and districts (3,131 to 3,970), 1 to 1.267, and that in the hospital (514 to 898), 1 to 1.75.¹

In 1799, the mortality was numerically and proportionally to the population, much smaller than it had been in former epidemic seasons. Not exceeding one thousand,² it bore a proportion to the population—which amounted to above sixty-five thousand—of 1 to 65. On reference, however, to the brief accounts handed down to us of this epidemic,³ it will be found, that the emigration was greater than it had ever been anteriorly; that it commenced even before the existence of the disease had been officially announced; and that by the first of September, ten days after that announcement, the number who had left was so great that extensive sections of the city and of Southwark had become almost depopulated. With this in view, we may easily perceive that, in proportion to those exposed, the mortality from the disease was probably as large in 1799 as it has proved in other sickly seasons. What the number of cases was, during the prevalence of the epidemic, and what the ratio of deaths to recoveries, we have no means of ascertaining. All we learn in regard to this latter point is, that out of an aggregate of three hundred and twenty-four cases sent to the City or Fever Hospital, one hundred and ninety-three died, presenting a ratio of 1 in 1.68. Of twenty-one cases, which occurred up to the 11th of July, eleven, or more

¹ See Preliminary Observations, pp. 86, 87.

² 1015—Mease, pp. 37, 38; Ruston, p. 64.

³ Currie, Fever of 1799, p. 24; Rush's Works, iv. 57.

than one-half, died; while in the Lazaretto or Marine Hospital, the loss—21 in 95—was at the rate of 1 in 4.52. Starting from these premises, and bearing in mind at the same time the results noticed at other periods, we cannot greatly err in estimating the mortality at 1 in 3, and the number of cases, consequently, at about three thousand.

The epidemics of 1802, 1803, and 1805, from a less degree of virulence and power of diffusion of the efficient cause, as well as from a greater restriction of the infected localities, aided doubtless by an early and extensive emigration, produced a smaller absolute mortality than had been noticed in former sickly seasons. Indeed, the loss compared with the population was, on these occasions, too small to afford us a subject worthy of consideration. Nor could we, were it otherwise, arrive at satisfactory results, owing to the impossibility, at the present day, of collecting information of sufficient precision to attain the object in view, especially as regards the two last mentioned seasons. In 1802, in reference to which our information is more ample, the number of deaths from the reigning fever amounted to three hundred and seven; in 1803, to one hundred and ninety-five; and, in 1805, to between three and four hundred. As epidemics, therefore, and taken in connection with the extent to which the population had grown, and even with the number of inhabitants who had remained in the city and districts, these visitations must be regarded as of very trifling importance.

As respects the number of cases that occurred, and the ratio of the mortality to the sick, in hospital and private practice, our information is not much more explicit. In 1802, the number of cases reported amounted to five hundred and ninety-eight, which—the deaths being three hundred and seven—gives us a ratio of nearly 1 in 2. In the hospital, the number of admissions was one hundred and ten, of which fifty-eight, or one-half, died. In 1803, eighty-seven cases were received into the hospital, where the deaths amounted to thirty-eight; being in the proportion of 1 to 2.3, or near one-half. But how the disease comported itself in that respect throughout the infected localities generally is not unknown, as we cannot ascertain the number of cases that occurred. Dr. Rush (vol. iv. p. 88) speaks of the mortality not having exceeded 5 in 100 in the practice of most of the physicians. Supposing this to be correct, the deaths having amounted, as stated, to one hundred and ninety-five, it would follow that the number of cases reached to three thousand nine hundred. But this computation is open to serious objections. Dr. Rush admits that the cases alluded to by him included all the grades and forms of the disease; and, as we know that he viewed in the light of true yellow fever complaints not usually recognized as such by other and experienced practitioners, the inference is natural that his estimate of the mortality can afford us no clue to a knowledge of the number of cases, and to the ratio of deaths from the former disease in the epidemic in question. Besides, the ratio of mortality given by him does not tally with that noticed in the hospital and in former and subsequent epidemic seasons, and would suffice to prevent us from placing full reliance, for the purpose before us, on the correctness of his statement. In the year 1805, the number of cases,

according to Dr. Caldwell, amounted to probably between twelve and fifteen hundred. Supposing it to have been thirteen hundred and fifty, and the deaths to have reached three hundred and fifty, we have a proportion of 1 in 3.86. On this point, however, there is no certainty. The hospital received three hundred and forty-six patients; but I am not able to ascertain the ratio of mortality among them.

During the next two epidemics by which the city of Philadelphia was visited—those of 1819 and 1820—the mortality was very small in proportion to the population, which then exceeded one hundred and eight thousand souls; owing, in the former season, to the circumscribed limits of the infected localities, and in the latter, to the hygienic means employed to arrest the progress of the disease. But, in proportion to the number of those attacked, the loss was unprecedentedly large. In the year 1819, the cases amounted to twenty-four, and the deaths to twenty, or 1 of the latter to 1.2 of the former. In 1820, one hundred and twenty-five cases were reported, and of these ninety-three terminated fatally, or 1 death in 1.5 cases. Of the mortality in 1853, it has already been stated that it amounted to one hundred and twenty-eight. The number of cases reported during the season did not exceed one hundred and seventy, which gives a proportion of 1.42, or 75 per cent. of deaths to cases.

If now we sum up the facts embraced in the foregoing statements, we shall find that, exclusive of the deaths that occurred from sporadic cases in 1796, 1806, '7, '8, and other years, and laying aside those seasons relative to which we have no precise information, the fever has caused a loss in Philadelphia, from 1699 to the present day, during eleven visitations, of not less than twelve thousand seven hundred and fifty-one individuals, being on the average of eleven hundred and fifty-five each sickly year. In the four epidemic seasons of 1793, '7, '8, '9, it caused a loss of ten thousand and thirty-eight. When we inquire into the ratio of mortality to the population, basing our calculations only on the information we possess relative to a limited number of years, we find it to have been 1 in 137.43 of the entire inhabitants, and 1 in 128.94 of those who remained exposed to the infection. The three epidemics of 1793, 1797, and 1798, alone give an average of 1 death in 14.24 of the entire population, and 1 in 10.03 of those that remained. The mortality among those affected, estimating it from the results recorded in each epidemic year, from 1793 downwards, varied from 1 in 1.2 (1819) to 1 in 3.86 (1805), giving an average for all those epidemics of 1 in 2.12. The loss at the hospital alone, during the epidemics of 1793, 1797, 1798, 1799, 1802, and 1803, the only years in which we find the admissions recorded, varied from 1 in 1.68 (1799) to 1 in 2 (1803), with an average for the six seasons of 1 in 1.867.

From these results, the conclusion is inevitable that the yellow fever, as often as it has appeared among us, has occasioned as large a mortality, in proportion to the whole population, or to that portion of it on which it more particularly exercised its action, as it has been found to produce in any part of the United States or elsewhere. Few cities beyond the tropics—New Orleans, Charleston, and one or two others excepted—have been as repeatedly visited

by the fever as Philadelphia was prior to 1820. In few has it spread more extensively than it did during some of our epidemics; and certainly in none, within the limits of this country, has it assumed a more malignant garb, and given rise to a greater ratio of mortality among those attacked. Nay, it may be doubted whether the records of West Indian or European epidemics exhibit more than a few instances in which the disease has proved more extensively destructive to human life. But it is to be borne in mind that the number of cases reported or estimated at each return of the disease in all probability falls far short of the true one. In all seasons, many—very many, indeed—of the mild ones are not taken heed of by their medical attendants, the health officers, or the public at large. They recover, and nothing is said about them. I know full well that such was the case in 1820 and in 1853, and there is no doubt that similar occurrences took place in former epidemics. Had all these cases been reported, and had our calculation of the mortality been based on the full amount of individuals affected, the result would have been less painful. But, after making every allowance possible for the number of such omissions, it is not to be concealed that the proportionate mortality has ever been considerable. Painful, therefore, as the avowal may be, every candid Philadelphian must admit that, much as some writers among us have glorified themselves for their extraordinary success in the management of the disease, the facts that have been laid before the reader justify the conclusion that, neither during our earlier nor our late epidemics, has anything been achieved, calculated to justify a claim on our part to superiority of success.

While saying this, I am far from attributing the great mortality in question to unskilfulness on the part of the physicians upon whom devolved the painful task of encountering the fever at its various returns. No one more than myself is sensible of their adequacy in that respect. Familiarity with their views—pathological and practical—relative to this formidable disease, derived from a study of their writings, from authentic tradition, from personal and intimate intercourse with a large portion of those who saw most of the disease, and from personal observation during the prevalence of two epidemics, as well as a full knowledge of the results obtained elsewhere, have left no doubt on my mind that they will bear comparison with the physicians of any other yellow fever region. Much more correct is it to refer the fatal results recorded to the deadly malignancy of the poison giving rise to the disease, and which, under particular circumstances, at once places a large number of those on whom it exercises its morbid influence beyond the reach of art. To any one conversant with the yellow fever, it need scarcely be told that in all epidemics cases occur in which the patient may be properly said to begin to die from the very outset of his attack; and that while, during some seasons, such instances, in which, do what we may, death is inevitable, are few—the majority of the cases being more or less tractable—epidemics occur in which they predominate; and others, again, in which they present themselves almost, or even to the entire exclusion of the milder forms. That the disease in some of our sickly seasons was of the latter deadly character; that in most of the others, if not all, it manifested the destructive tendency adverted to, in

a very large number of those attacked, every one must know who has enjoyed the advantages of personal observation, or has perused attentively the descriptions of it handed down to us by our predecessors. In no season has the fever assumed the benign form which enables the large majority of patients to get well under a mild treatment, or even without any treatment at all. The occurrence has been noticed elsewhere. In the epidemic of 1839, at New Orleans, the disease, in the greater number of the cases, though exhibiting its characteristic phenomena, was of the kind mentioned, and consisted of little more than the first stage, which, when subsiding, left the patient in a state of convalescence. Much the same results were obtained in that city in 1847 and 1849; at Woodville in 1844, Natchez in 1848, and Mobile in 1847; in Bermuda in 1819; and in the Havana in 1837. "It has been expressed to me," says Dr. E. H. Barton, of New Orleans, "by some of our oldest inhabitants, those who have been observers of the disease twenty or thirty years ago, that it was no longer the same disease, that, in fact, the unequivocal malignancy and peculiar type which characterized it exist no longer; and this is most amply verified by the symptoms, aspect, and history of the disease in its various stages, as seen and described by those who had witnessed it from 1804 to 1823;" "and that, for some years back, it has blended itself with the ordinary diseases of the country." "Every now and then we nevertheless meet with cases where there is no room for doubt, but they bear a very small proportion to the mass of cases which occur here every year."¹ This, however, has never occurred in Philadelphia, where, as exhibited by the mortality it occasioned, the disease has invariably been marked by intense malignancy. To this the epidemic of 1803 cannot be viewed as forming an exception; for, owing to reasons already assigned, the proof that might be derived from the statement of Dr. Rush, respecting the ratio of mortality in that season, is vitiated by the admission that under the name of yellow fever were included all the forms and grades of febrile complaints treated during the whole sickly period.

From all this it is evident that, under circumstances of so unpromising a kind as those noticed here at every occurrence of the fever, it would have been impossible to obtain a success different from that recorded in the preceding pages; and which, though greatly to be deplored, and affording no ground for the self-glorification in which some of the physicians of former days indulged, is nevertheless not inferior to that frequently noticed in other parts of the United States, and in foreign countries.

From the history of the few epidemics of this city of which proper details have been preserved, we perceive that the mortality has varied, numerically, at each return of the disease, during the several months of its prevalence. In 1793, the disease caused the greatest loss in October, when the mortality amounted to little less than two thousand (1,996)—September furnishing one thousand four hundred and forty-two. The extremes in the former month were 13 (on the 27th) and 119 (on the 11th). The same results obtained in

¹ Report to the Louisiana State Medical Society, p. 30.

1802, when October furnished the largest number of fatal cases. On the other hand, in the years 1699, 1762, 1797, 1798 and 1799, the mortality attained its culminating point in September. In 1798, the number of deaths during this month exceeded, by nearly four hundred, that in August, October and November combined; while in the other epidemics mentioned, the contrast between that and the other months, though not so excessive, was still very considerable.

Nor is it less certain that, in our various epidemics, the mortality has differed greatly, both as regards the amount of the deaths and the ratio of these to the number of cases, in the several portions of the city where it prevailed. This was observed in 1793, and is recorded by Dr. Barnwell (p. 374), Currie and others. Dr. Rush points out the same circumstance in reference to 1797, 1802, and 1803.¹ Of the latter epidemic, Dr. Caldwell remarks: "As the fever receded from the low ground and malignant atmosphere of Water Street, it became more and more manageable."² Of 1802, Dr. Rush says: "Nearly all the persons who were affected with the fever in the neighbourhood of Vine and Water Streets, and in Walnut and Spruce Streets, died" (iv. 88). A similar observation was made in 1805; the fatality of the fever being proportionate to the distance from the river (Caldwell, p. 51). The fever of 1820 furnished a striking example of the difference in the proportion of deaths to recoveries in several localities. While at Walnut Street wharves and the vicinity nearly one-half of those attacked recovered, and in Front Street below Walnut, and in Lætitia Court, the recoveries were more numerous, three-fourths of the cases at Hodge's wharf and vicinity, and almost all those in the Northern Liberties, ended fatally. The ratios stood as follows: 9 to 4—25 to 22—5 to 3—9 to 2—1 to 3—2 to 2—1 to 3—3 to 2—11 to 1. Dr. Jackson, in his account of this epidemic, while alluding to the fact in question, says: "Dr. Monges, whose accurate knowledge of this disease is so well known and acknowledged, states that he has noticed the same facts in the different epidemics of our city. In certain situations, when the disease was general, he found it light and readily manageable; all his patients recovered; in other situations, it was precisely the reverse; medicine was unavailing, and his patients nearly all died" (p. 62).

The rate of mortality from the disease in this city has invariably varied at different periods of the same epidemic. Dr. Chapman,³ who, like most other writers on the same subject, has viewed this as one of the laws of epidemics, says of the yellow fever: "Always in the beginning mortal in nearly every instance—progressively more tractable—till finally it becomes comparatively curable." Of the fever of 1793, Barnwell states, that from its first beginning the disease exhibited something extraordinary in the mortality and rapidity of its progress, but towards the close of October and November, it became less dangerous and rapid (p. 388). In 1798, while the loss in the city during

¹ Vol. iv. pp. 85, 88.

² See Med. Repos., vii. 149, 188; Caldwell, Med. and Ph. Mem., p. 112.

³ Chapman on Epidemics, Philad. Med. and Phys. Journal, ix. 12, 107.

the whole season was 1 in 1.34, the ratio in August was 1 in 1.32; or 1 in 1.56, if we add to the number of cases the one hundred and fifty that occurred prior to the 9th. In September, the ratio was 1 in 1.143, and in October and November, 1 in 0.908. In the hospital, the ratios were 1 in 1.2, 1 in 1.75, and 1 in 1.53. The same difference in the ratio of mortality was observed in 1803 and 1805,¹ when the disease became less fatal as the season advanced. This law, however—if law it is—is subject, like every other, to exceptions, when the disease manifests itself in its most fatal garb at the close, instead of the outset, of the epidemic. In the year 1820, the worst and most fatal cases occurred in October; out of twelve reported in a circumscribed locality, eleven terminated fatally.

The diffusion and fatal tendencies of the disease in this city may well be compared to those of other diseases of kindred and equally malignant character. The fever which swept over the Coromandel in 1809, 1810, and 1811, caused the loss of 106,789 out of a population of 1,828,610. Coimbatore lost in sixteen months 22,451 out of 596,606; Madura, in twelve months, lost 24,626 out of 245,654; Dendigul, in the same space of time, had 21,510 deaths in 295,654 individuals; while at Tinnivelly, the mortality, in the short space of five months, exceeded 38,202 in a population of 690,696.²

In addition to this, the attention of the reader, curious in these matters, may be called to the wide diffusion and excessive mortality noted during some epidemics of glandular plague. He may be reminded that, in London, in 1625, it carried off not less than 35,417 individuals, according to Grant,³ and 46,000, according to the calculation of Short;⁴ that, in the same city, the mortality, forty years after (1665), amounted to 97,000,⁵ in a population of less than 500,000; that, in 1812, the loss at Constantinople amounted to 159,534;⁶ and that in Lyons, in 1628, '29, in a population of some 200,000,⁷ fifty thousand persons were destroyed by the same disease. He may also be told that, at Montpellier, the number of deaths in 1629 fell but little short of one-half of the inhabitants who had not left the city;⁸ that, in Marseilles, the loss from the memorable pestilence of 1720, amounted to forty thousand;⁹ that at Noja, in 1815, '16, the number of cases in a population of 5,300 reached 1,474, and the deaths 716;¹⁰ that at Cyprus, in 1760, the disease destroyed 70,000, out of a population of 600,000;¹¹ again, that at Aleppo, the population of which amounted to some 150 or 200,000, it carried off in two years (1761, '62) about 21,800;¹² that in the plague of the

¹ Med. Repos., vii. 188; Caldwell on Fever of 1805, p. 51.

² Med. Geog. and Agricult. Rep. on Fever of Coimbatore, &c., by Ainslie, Smith, and Christy. Lond. 1816, p. 93, &c.

³ Natural and Political Observations, &c., made upon the Bills of Mortality, p. 8.

⁴ New Obs. on Bills of Mortality, p. 274.

⁵ Short, op. cit., p. 292; Marshall, Stat. and Mortality of the Metropolis, p. 66.

⁶ Brayer, Neuf. Années à Constantinople, ii. 248.

⁷ Papon, De la Peste, Ou les Epoques Mémoires de ce Fléau, i. 184.

⁸ Ibid., p. 195.

⁹ Ibid., p. 343.

¹⁰ Moreo, Storia della Peste de Noja, p. 25; Tavola, 3.

¹¹ Russell on the Plague, p. 8.

¹² Ibid.

same city, which immediately preceded the year 1797, about 60,000 inhabitants were swept off,¹ and that, in 1835, the loss in Cairo was little short of 26,000.² But it is unnecessary to pursue the subject.

Before bringing these remarks to a close, it may not be uninteresting to examine how far the yellow fever of Philadelphia assimilates itself, in respect to the loss of life it has occasioned, and the ratio of its mortality to recoveries, with the same disease as observed in other parts of this country, in Europe, and in tropical climates. In doing so, reference will be made to all the facts bearing on the subject to which I have had access. But, while collecting information relative to the mortality of the fever, from all sources within my reach, both domestic and foreign, I am obliged to exclude from the list some statements which have, at different times, been laid before the medical profession. Allusion is here more particularly made to those contained in an *Essay on the Yellow Fever*, by Dr. John Hastings, late of the United States Navy, and in which the loss from the fever, amid a very large number of cases, is represented as so small and trifling, as to justify the suspicion that the writer has had principally to deal with a disease different from that which forms the subject of our present inquiries. According to Dr. Hastings, in the epidemic of Tabasco, on the coast of Mexico, where the fever from which the American sailors suffered so much, during the late war, first commenced, there occurred "considerably more than three hundred cases." Of these not one died. On board of the Mississippi frigate, during the same summer, "more than four hundred cases of the fever occurred during the months of July and August." The loss was two men. At the Hospital of Salmadina, over four hundred cases were treated. Of those received within the first forty-eight hours, four died; while the loss, among the cases admitted from the fourth to the seventh day, amounted to nine—in all thirteen. All the fever cases landed, in 1841, from the U. S. brig Jefferson, at the Hospital of Indian Key, within the first three days of the attack, recovered. The same success attended among the sick who landed from the schooner Otsego. The vessel was sent back to the west coast with a fresh crew, officers and men, "but in a short time again returned, to land all hands, down with the fever." They all recovered. "Thus," says Dr. Hastings, "the result of the treatment as applied to the disease in the several times heretofore related, was the loss of half per cent. in those received in the early stages of the attack." When we find a disease producing a mortality of such trifling extent—so much smaller than that occasioned by our ordinary remittent; when we compare this mortality with that ordinarily noticed in every yellow fever locality, and under every possible mode of treatment; when we learn that this success has been obtained by remedial agents once highly eulogized, but long since abandoned everywhere as of more than suspicious efficacy, and when we discover that this treatment is predicated on the supposition that the disease depends on violent inflammation of the brain and its

¹ Encycl. Britan., ii. 400.

² Boudin (from Gaetani Bey), Geogr. Med., p. 14.

envelops; we cannot greatly err in doubting the propriety of appealing to those statements as illustrative of the mortality, under certain circumstances, of true yellow fever, to say nothing of the fact that the description by Dr. H. of the disease which fell under his observation, does not exactly correspond with that we possess of the fever in question, as it has appeared here and elsewhere.

Equal suspicion may be attached to the statements of other physicians. Lefoulon informs us that, in the space of some fifteen months, he treated four hundred cases. Of this number, four died. This certainly was but a very small loss. But the success obtained will appear still more extraordinary when we find that two of those who died, did so from a relapse, and another, from the use of improper remedies taken clandestinely.¹ From this, it follows that Lefoulon cannot fairly be said to have lost more than one patient out of four hundred. This gives a proportion of a quarter per cent., a result which is superior to that of Dr. Hastings. In Currie's *Reports*, we find the copy of a letter from a Mr. Nagle, surgeon of the ship *Royal Sovereign*, who states that while on board of the *Ganges*, on the Jamaica station, he treated one hundred and twenty cases of yellow fever. Of these, two died.² Mention is also made of a Mr. Wilson, of the ship *Hussar*, who had eighty-three cases under his care, and did not lose one.³ Dr. Barrington, of the U. S. Navy, in a publication on the yellow fever, to which I have already had frequent occasion to refer, says: "I do not mean to question the veracity or accuracy of Professor Potter, of Baltimore, when, in his lectures on yellow fever, in relating the success of his practice in three different years, he states that in the first year, out of seventy-two patients, only three died; in the second year, out of four hundred, only five died; and in the third year, out of forty-eight, not one died." Dr. Barrington asks: "Who has ever boasted of anything like such success? May it not be asked, were they all cases of yellow fever?"⁴ He had not consulted Lefoulon or Currie, and Dr. Hastings had not yet written. Another and more recent example of extraordinary success presents itself, and deserves to be recorded. Dr. Walkly, of Mobile, informs us, that during the epidemic which prevailed in that city in 1853, he treated, between the 12th of August and the 1st of November, three hundred and thirty-six patients consecutively, and that out of that number ten died. This gives us a mortality of a little less than three per cent., or 1 in 33.6.⁵

It may not be improper to remark that in all these instances of unprecedented success, the results were obtained by modes of treatment of the most opposite kind. Dr. Hastings trusted to ample depletion, general and local, and mercury. When used early and boldly, these means never failed to remove the disease as if by magic. Dr. Potter had recourse to calo-

¹ F. Adynamiques, p. 7.

² Med. Reports on the Effects of Water, ii. 137-8; Edinb. J., vii. 20.

³ Ibid., ii. 216-17; Edinb. J., vii. 20; Trotter, Med. Naut., i. 361.

⁴ Am. J., xii. 317.

⁵ N. O. J., xi. 293.

mel in scruple doses every two hours, with a view to excite salivation. Le-foulon employed neither mercury nor the lancet, but resorted in the early stage to strong emetics followed immediately by Peruvian bark and stimulants. Mr. Nagle was satisfied with the effects of cold affusions, which, "when used in the first, or even second day of fever, operated like a charm." Mr. Wilson employed the same remedy, but also bled and gave a solution of emetic tartar. Dr. Walkly is the advocate of a different mode of treatment; neither bleeding, leeching, mercury, quinine, nor cold affusions. An emetic in the commencement—next a mild purgative, or injection, a hot mustard foot-bath, and stimulants when symptoms of prostration supervened, constituted the principal means resorted to by him. Certainly, the yellow fever must be an extraordinary disease, to yield, in the hands of some physicians, as readily as a common cold—and that, too, when opposed by such diversified means—while in the practice of other and equally expert physicians, in this and other countries, it proves so rebellious to the same means as to carry off a very large amount of the sick, and, indeed, to appear to be, by some of them, rendered more readily fatal.

If, while bearing in mind that the loss in this city has varied in the several epidemics from 1 in 1.2 to 1 in 3.86, giving an average of 1 in 2.53, the reader examines the following tabular view of the results obtained in the fever of tropical climates, he will perceive that, excepting in a few instances, when the disease would seem to have assumed an unusually mild character, or to have prevailed, as at Boa Vista, among individuals who, from constitutional peculiarities, or the effects of acclimatization, are little liable to the severe forms of fever; or when it has presented itself in a garb calculated to justify doubts as to its genuineness, the mortality there, though sometimes exceeding, and occasionally falling short of, that experienced here, more frequently equals it; while the average of the ratios, furnished by a series of epidemic seasons, approximates very closely in both regions. Omitting the instances of an exceptional kind referred to, those extremes will be found to vary from 1 in 1.08, to 1 in 10; the average of all the ratios being 1 in 2.32. The yellow fever of this city, therefore, though sometimes less fatal than that of tropical climates, has upon several occasions proved more so than is usually found to be the case there. As regards the proportion of the mortality to the population of places visited by the disease in those latter regions, nothing need be said; as the larger number of the residents of such places, being natives or acclimated foreigners, are placed beyond the risk of an attack, and no record is kept of the number of susceptible persons.

I. TROPICAL CLIMATES.

AUTHORITIES.	PLACES.	POPULATION.	NUMBER OF CASES.	MOR- TALITY.	PROPORTION TO POPU- LATION.	PROPORTION TO CASES.
Rochoux (1828), p. 568.	West Indies generally.	1 in 1.5 to 1 in 1.33.
Robert Jackson, Sketch, p. 13.	"	1 in 3, often 1 in 2.
Proudfoot, Edinb. Journ., pp. 28, 295.	"	1 in 2	1 in 2.34.
Statist. Rept. of British Army, p. 50.	Windward and Leeward Islands.	1 in 1.5.
Chisholm, i. 451.	" " Sir Ch. Grey's Army, 1794.	12,000 (estim'd)	...	6,012	1 in 2	1 in 1.33—1 in 1.2.
	{ St. Domingo, 1734.	1 in 2	
	" 1802.	40,000 (soldiers)	27,000 (estimated)	20,000	1 in 2	
	" 1798.	25,000 (soldiers)	1 in 1.14	
	" Port Mirbalais.	300	...	290	1 in 1.04	
	" St. Nicholas, 1796.	1 in 2.
	" Port au Prince }	1 in 2.
	" (Rohan Hussar). }	1 in 2.
	" St. Mark.	500 (troops)	200	100	1 in 5	1 in 2.
	Demerara, Georgetown Hospital.	...	{ Severe, 2071 } 3032	404	...	{ Severe, 1 in 4.54
	{ Martinique, 1823-7 (Hospital).	...	{ Mild, 961 }	{ All, 1 in 7.5.
	" 1838-9.	{ 1 in 2.5, 1 in 3,
	" first 3 mos. of 1839.	...	1,344	223	...	{ 1 in 3.5, 1 in 1.5.
	" Oct. 1, 1838, to }	...	92	19	...	1 in 6.
	Sept. 30, 1839. }	...	1,202	150	...	1 in 4.5.
	" 1818.	...	1,982	697	...	1 in 8.
	" 1825.	...	1,464	388	...	1 in 2.82.
	" 1821.	...	686	235	...	1 in 3.8.
	" 1802-7 (soldiers).	11,085	8,673 (estimated)	2,891	1 in 3.8	1 in 3.
	" By Dariste, from Aug. }	...	327	61	...	1 in 5.36.
	" 1816, to close of 1817. }	1 in 4.5.
	" last 6 mos. of 1803, and }	...	2,462	546	...	
	first 6 mos. of 1804. }	727	1 in 17.34	
	" 1852, Port Royal.	12,611	...	1,200	1 in 17	
	" St. Pierre.	20,360	

Rochoux (1828), p. 568.
Robert Jackson, Sketch, p. 13.
Proudfoot, Edinb. Journ., pp. 28, 295.
Statist. Rept. of British Army, p. 50.
Chisholm, i. 451.

Desportes, i. 55; Gilbert, p. 70.
Bally, pp. 80, 286; Caillot, p. 160.
H. McLean, R. Jackson's Outline, pp.
61-2-4.
R. Jackson on Cold Affusions, pp. 230-31.

Blair, p. 49.

Catel, pp. 14, 20, 21; Rufz, p. 10.
Lefort, Du Quinquina, et de la Saignée,
p. 586.
Kéraudren, pp. 14, 15.
Moreau de Jonnés, p. 349.
Dariste, p. 24.
Edinb. Journ., lxxix. 236.

Kéraudren, pp. 14, 15; Bally, p. 81, 287. Caillot, p. 170; Vatable, p. 347. Chisholm, pref. 2d ed., p. xvii. Chisholm, i. 451; ii. 119. Arnoux, An. Marit., 1844, ii. 758. Cornuel, An. Marit., 1844, ii. 735. Moreau de Jonnés, p. 349.	{	Guadaloupe, 2 Point à Pitre.	3,500 (troops)	...	475	...	152	...	1 in 3.1.
	{	"	1803.	...	3,700	2,700	1 in 1.3	...	
	{	"	1826.	...	2,757	...	386	2,900	...	1 in 1.21.	
	{	"	1843 (sailors and troops).	772	128	...	1 in 3.	
	{	"	Basseterre.	96	183	1 in 15.06	1 in 4.22.	
	{	"	" 1833 (soldiers).	137	64	...	1 in 15.	
	{	"	1796-9.	...	20,000	47	
	{	"	1796.	...	367 (artillery)	13,807	1 in 1.47	...	
	{	"	1802-7, inclusive.	...	16,363	129	1 in 2.8	...	
	{	St. Lucia, 1664-6.	...	1,500 (soldiers)	...	500 (sailors)	5,057	1 in 3.2	...
Father Du Tertre, iii. 86, 244. Chisholm, i. 124. Dr. John Hunter, p. 47.	{	"	...	5,000	1,411	1 in 1.06	...	
	{	Grenada, 1793 (sailors).	200	1 in 2.5	...	
	{	" sailors, soldiers, and white inhabitants.	{ Nearly all in 1 year	
	{	" white inhabitants.	1 in 3.	
	{	"	1 in 7.	
	{	Jamaica, 1815.	1,130 (soldiers)	1 in 5.	
	{	" 1819, '21, '27.	630	1 in 4.	
	{	" 1819, '22, '25, '27.	{ 1 in 2, 1 in 4, 1 in 1.08.	
	{	" 1819.	2 regiments	1 in 2.	
	{	" Stony Hill, 1827.	300	1 in 2, 1 in 1.7.	
Statistical Reports, pp. 46, 94. Arnold, pp. 148, 149, 165, 171, 247.											
In 1802 the mortality among troops amounted to 60 per cent.											
1802 1803 1804 1805											
Mortality in West Indies among French troops.											
1802 1803 1804											
Marshall, Sketch of the Geogr. Distribution of Diseases, Edinb. Med. and Surg. Journ., xxxviii. 345.											

I. TROPICAL CLIMATES—CONTINUED.

AUTHORITIES.	PLACES.	POPULATION.	NUMBER OF CASES.	MOR- TALITY.	PROPORTION TO POPU- LATION.	PROPORTION TO CASES.
Moseley, p. 163.	San Juan.	1,800	...	1,420	1 in 1.3	1 in 3.58.
Proudfoot, Edinb. Journ., xxviii. 295.	St. Christopher, 1812.	...	422	118	...	1 in 13.
Jones, Lond. Med. Repos., March, 1823.	Bermuda, 1819.	...	208	32	...	1 in 4.33.
	Barbadoes, 1816.	...	{ 390 men 25 officers	90	...	1 in 2.5.
Proudfoot, Edinb. Journ., xxviii. 295.	" 1820.	10	...	1 in 2.56.
Ralph, Edinb. Med.-Chir. Trans., ii. 61.	" 1838.	1 in 4.25.
J. Davy, Notes on Blair, 49.	" 1841.	1 in 1.2.
Finlay p. 25.	" 1842.	1 in 5.6.
	" 1852 (troops).	1 in 5.08.
	{ Havana, 1837.	1,380	879	173	1 in 7.9	1 in 10.
Maher, Ann. Maritimes, p. 878.	{ " Belot's Hospital.	1 in 6.48.
Holliday, pp. 9, 10.	" 1794.	{ 1 in 1.1 in some ships	1 in 1.1.
Imray, Edinb. Med. Journ., lii. 95.	{ Dominica, 1838.	131 (soldiers)	{ 100 men 6 officers	35 men	1 in 3.72	{ 1 in 3 men. 1 in 2 officers.
Ibid., lxiv. 328.	" 1841.	...	204	55	...	1 in 3.7.
Tullock's Reports, p. 19.	Trinidad, 1818.	1 in 2.54.
Humboldt's New Spain, p. 777.	{ Vera Cruz, 1803 (hospital).	16,000 to 17,000	428	69	1 in 2.40	1 in 6.2.
Ibid., p. 779.	" in 15 years.	{ 1 in 2.8 to 1 in 6.23.
Humboldt, Personal Narrative, iii.	Porto Cabello.	1 in 1.54.
	{ Boa Vista, 1845, Porto Sal Rey.	Portuguese, 53	47	25	1 in 2.1	1 in 1.8.
	" " "	Eng. and Am., 11	8	7	1 in 1.6	1 in 1.1.
	" " "	Natives, 915	...	68	...	1 in 13.4.
McWilliams's Report, pp. 87, 88, 91.	" in all localities.	Europeans, 69	...	32	...	1 in 1.16.
Med.-Chir. Rev. for July, 1847, pp. 233-4.	" " "	Natives, 4,309	...	279	...	1 in 15.4.
	{ Georgetown (Demerara) Sea- man's Hospital, in 1838, &c. }	...	2,071	404	...	1 in 5.12.
Blair, p. 49.	Cayenne, 1850 (Hospital).	...	685	148	...	1 in 4.63.
Report by Drs. Ginoaves, Reboue, &c., Ann. Marit., 1852, viii. 179.						

Passing from the fever of tropical climates to that of Europe, as exhibited in the annexed table, we find that the reverse is the case; for while the fever there has occasionally produced a smaller degree of mortality than it has usually done here, and has at other times approximated closely to our disease, it has more frequently than in the West Indies given rise to a larger amount of deaths. It will be found, also, that, during a more limited number of epidemics, some European cities have exhibited a larger diffusion of cases, and experienced a greater loss of life, both numerically and in proportion to the amount of the population. Spain, the principal sufferer on occasions of the kind, experienced, in the course of eight epidemics—1800, 1801, 1803, 1804, 1810, 1813, 1819, 1821—the immense loss, by the fever, of upwards of 130,000 individuals. Barcelona and Cadiz alone have suffered a loss, during single epidemics, nearly equal to that which Philadelphia sustained in all her sickly seasons combined. During one single season—that of 1800—not less than thirty cities, towns, and places of Andalusia were visited, and in them the loss amounted, according to the computation of Alfonzo de Maria,¹ to 61,363, distributed as follows:—

Seville	14,685	Carraca	515
Cadiz	10,986	Coria	450
Xerez	10,192	Espera	442
Isla de Leon . . .	5,033	Los Palacios . . .	192
Puerto de Sta. Maria .	3,695	El Arahall	180
Sanlucar	2,303	La Carlota	147
Lebrija	2,100	Medina, Sidonia . .	136
Moron	1,854	Paterna	86
Utrera	1,689	Dos Hermanas . . .	70
Puerto Real	1,621	Tribujena	68
Chiclana	1,328	Bornos	17
Rota	1,116	Mairena	9
Las Cavezas	994	Sara	5
Alcalà de los Gazules .	817	Estepa	2
Arcos	631	Vellamartin	1

The year 1804 was another of great calamity. On that occasion, twenty-five cities or towns were severely visited by the fever. The population in these amounted to 427,228, of which 52,559, or 1 in 8.12, perished. In fourteen of these places, at different periods, the mortality in proportion to the population was 1 in 6.42, the extremes being 1 in 2.25 and 1 in 13.3. In seven places, the proportion of persons affected amounted to 1 in 2.78 of the population, the extremes being 1 in 1.18 and 1 in 5. In twenty-one, the average proportion of deaths to the number affected was 1 in 3.087, the extremes being 1 in 1.3 and 1 in 6.42; while ten hospitals gave a mortality of 1 in 2.15 of the number admitted, with extremes of 1 in 1.1 and 1 in 3.82. The whole number of items noted in the table gives an average of 1 in 3.55, the extremes being 1 in 1.1 and 1 in 9.6.

The imperfect character of the documents we possess relative to the mor-

¹ *Memorie sobre la Epidemia de Andalusia, &c.*, p. 122.

tality from the yellow fever of Europe, renders it difficult to compare the ratios in the several epidemics by which separate cities or towns have been visited with those noticed here; for, though some of those localities have been several times the seats of considerable devastation from the disease, we fail in our endeavours to ascertain the results obtained in any of them during more than one, or, at most, two epidemic seasons, and but too frequently they come to us in a shape well calculated to make us suspicious of their accuracy. The city of Cadiz, where the number of cases reported has always been very large, present us with a ratio at one time of 1 in 6, and at another time, of 1 in 9—a success which has never been obtained here. But as the hospital patients, on the same occasions, died at the rate of 1 in 2; and as the mortality in other cities of Spain, during the same seasons, was very large; and as, moreover, the Spanish physicians are not supposed to be strong on the score of diagnosis, it is more than probable that many of the cases reported as yellow fever, and which contributed to swell the list of recoveries, belonged to a different category of febrile diseases, and not to the one which forms the subject of our present inquiries. In Seville, though the loss on one occasion was smaller than it was with us, it approximated closely to our ratio at subsequent periods. The same thing may be said of Xerez. In Gibraltar, the fever on two occasions approximated to our less fatal visitations; and in Barcelona, Malaga, Carlotta, &c., it approached our worst.

<p> Pariset, pp. 55, 435. Chervin, Examen., p. 202. Pariset, Obs., p. 64. Med. Statist. of British Army, p. 8. Gilpin, Med.-Chir. Trans., v. 337. Edinb. Med. Journ., x. 317. Fellowes, pp. 76, 449, 50, 1-3. Pym, p. 26, 2d ed.; pp. 47, 250, 1st ed. Hennen's Med. Topog. of Medit., pp. 98, 113. Proudfoot, Edinb. Med. Journ., xxviii. 295. Louis, p. 259; Smith, Edinb. Journ., xxxv. 41. </p>	<p> Tortosa, 1821. Porta Reale. Gibraltar, 1804. (NOTE 3.) " Hospital. " 1813. " Hospital, 1814. " civilians. " 1828. " troops. " civilians. Barcelona, 1803. " 1821. " Seminary Hospital. " General Hospital. " Marine Hospital. " Lazaretto of V. " Queen of Peru. " city and suburb, according to Audouard. Palma, 1821. Port du Passage, 1823. Leghorn, 1804. " Hospital. </p>	<p> { 15,000, remaining 5,000 } 20,000 10,000 estimated { 20,501, remaining 12,501 } 20,652 3,652 17,000 { 145,000, remaining 70,000 } 12,000 remaining { 3,000, 1,200 remaining } { 60,000, 48,000 remaining } ... </p>	<p> ... 5,500 ... 2,754 2,847⁹ 726 ... 6,715 2,014 4,701 73 14,000 1,739 830 79 56 20,625 7,400 85 101 (Audouard) ... 164 </p>	<p> 2,356 688 5,946 894 904 114¹⁰ 132 1,796 515 1,281 30 9,730 1,265 749 55 39 16 to 17,000 5,341 24 40 Audouard 655 56 </p>	<p> { 1 in 6.4 of entire } { 1 in 2.1 of remaining } 1 in 29.07 1 in 2 ... { 1 in 22.67 of entire } { 1 in 13.83 of rem'g } ... 1 in 11.5 1 in 8.41 1 in 13.3 ... 1 in 15, or 1 in 7 1 in 8.8, 1 in 4.24 1 in 1.62 1 in 125, or 1 in 50 1 in 75, or 1 in 30 </p>	<p> 1 in 9. 1 in 3.1. 1 in 3.4. 1 in 6.36. 1 in 3.73. 1 in 3.91. 1 in 3.6. 1 in 2.43. 1 in 1.33. 1 in 1.3. 1 in 1.1. 1 in 1.44. 1 in 1.44. 1 in 1.25. 1 in 1.5. 1 in 3.96. 1 in 2.5. 1 in 3. </p>
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¹ According to Alfonso de Maria, the mortality was 10,186, p. 122.

³ According to Bally. According to Alf. de Maria, 3,200, p. 122.

⁵ Dumeril says 20,000.

⁷ Alf. de Maria, 3,693, p. 122.

⁹ From 8th September to 3d December.

² According to Dumeril.

⁴ Alf. de Maria, 2,239.

Bally says 14,000. Alf. de Maria, 10,192.

Ibid., 2,303, p. 122.

1,400, according to Hennen, p. 113.

NOTES.

NOTE 1.—Seville, 1800. When we read the statement here recorded of the diffusion and ratio of mortality of the disease on that occasion, authentic as it appears to be, we can with difficulty divest ourselves of the idea of error having crept in somewhere. Not that I feel disposed to swell the mortality to 20,000, as was done by Dumeril (Humboldt, 779), and thereby diminish the ratio of recoveries; but I am inclined to regard it as probable that the number of those stricken down with the real malignant yellow fever did not reach the amount mentioned. The idea of an epidemic extending its baneful influence to all but 4,000 in a population of 80,000, is difficult to reconcile with the facts known respecting the diffusion of the same disease elsewhere. More natural is it to presume that in the number of cases recorded are included many of other and milder complaints. Should this be true, the proportion of deaths to recoveries would be larger than stated; while the number of cases might still be very large in proportion to the population. To this opinion I am the more inclined, because, so far as we can ascertain, the Spanish physicians, of those times at least, were not noted for accuracy in matters of diagnosis, and their success in the treatment of the disease has not usually been such as to induce the belief that they would lose but 1 in 5.21 during a wide-spreading and highly malignant epidemic. The same remarks are equally applicable to the reported occurrences in Cadiz and other cities of Spain.

NOTE 2.—“The mortality from the yellow fever has been prodigiously great in most of the seasons in which it has appeared in Andalusia; but the precise proportion of deaths to recoveries is not correctly known, as the official reports are not made up with exactness. It amounted to 70 in 100 at Xeres de la Frontera, according to the official return. It was in reality much higher, for those only were included in the dead list who were buried without the town. Private burial was obtained for many, and such were not registered, at least not correctly. Many, even a great many, were entered on the yellow fever list by connivance—that is, for the sake of the allowance granted by the municipality for that description of sick only. The diseases of those so entered were, it may be presumed, of a less mortal character than the yellow fever; consequently, the real truth is disguised in such a manner that the public is deceived, and the medical faculty can form no accurate conclusion. It may be said, with safety, that the mortality was not in reality less than one-half of those who were attacked.”—*R. Jackson, M. D., Remarks on the Ep. Yel. Fev. of the Coast of Spain*, pp. 137–8.

NOTE 3.—Dr. (now Sir) Joseph D. Gilpin reports the deaths from the 30th of September to the 1st of December, soldiers and civilians, at 6,524. He does this on the authority of a very respectable inhabitant of the place. But as he was not in the garrison at that period, he cannot vouch for the accuracy of the report. This latter includes 730 deaths among the Jews, which are not mentioned in the official report published in Sir James Fellowes and Sir W. Pym's works, which I have used.¹

The number of cases is not stated. Dr. Proudfoot says that the mortality was more than 1 in 3, and that only 28 persons escaped an attack out of 9,000 inhabitants. If such was the fact, the mortality was 1 in 1.5, as the cases must have amounted to 8,972, which, divided by 5,946—the number of deaths reported—gives the ratio mentioned.²

The disease, as it has appeared in various parts of the United States—east, north, south, and southwest of Philadelphia—presents results differing but little, in ordinary seasons, from those to which reference has already been made. On this subject, however, the documents within our reach are far

¹ Med.-Chir. Trans., v. 337.

² Edinb. Med. and Surg. Journ., xxviii. 295.

from being as ample and satisfactory as might be desired, with a view to arrive at precise conclusions; the statement of the mortality, as was often the case here, being frequently unaccompanied by a report of the number of cases. The extremes of the ratios, varying, as they do, from 1 in 1.5 to 1 in 13.1, are more widely separated from each other than those we have noticed in this city. It is to be remarked, however, that in all the epidemics in which the mortality has been set down as 1 in 9, 10, 11, 12, or 13, the disease is acknowledged to have been of an extremely mild character, and, as may be easily perceived from the descriptions extant, evidently assumed a form never seen among us, or indeed in any of our neighbouring cities. Under all other circumstances, the ratios approximate very closely to ours, whether the fever prevailed in places where mild epidemics sometimes occur, or in others where they have never been seen. In hospital practice, the results approximate closely to those obtained here, varying, as they do, from 1 in 1.6 to 1 in 3.15, with an average of 1 in 2.23. Excluding the exceptional seasons adverted to, the average ratio of all the statements, including those relative to the mortality in public institutions, is 1 in 2.53, the extremes being 1 in 1.2 and 1 in 6. Leaving aside the latter, we have an average of 1 in 3.5, and including the whole series, 1 in 3.38.

Except as regards a few of the places in our Middle and Northeastern States, where the fever has occurred, no advantage can accrue from comparing the mortality with the entire population, or with that portion of it that may have remained exposed during the continuance of the epidemic; for in most of the cities or towns liable to be visited by the disease—those of our Southern States—the greater number of the inhabitants are, as those of tropical climates, exempt from the disease, through the effect of acclimatization obtained by nativity, prolonged residence, or previous attacks; and the only class liable to infection is composed of strangers, the number of whom it is difficult, if not impossible, to ascertain.

III. UNITED STATES.

AUTHORITIES.	PLACES.	POPULATION.	NUMBER OF CASES.	MORTALITY.	PROPORTION TO POPULATION.	PROPORTION TO CASES.
Bayley, p. 91; Hardie, pp. 52, 143.	New York, 1795.	54,000	800	1 in 67.5	1 in 2.59.
Hardie, Fever of 1822, pp. 6, 7, 12, 18; Med. Rep., ii. 325, iii. 271.	" 1796.	...	178	69	...	{ In Aug. 1 in 2, afterw'ds 1 in 3.
Bayley's Letters from Board of Health, p. 40.	" 1798.	2,450	...	1 in 1.9.
Report on Quarantine, pp. 9, 10.	" Bellevue Hospital.	...	389	205	...	1 in 2.7.
Report of Board of Health of 1806, p. 13.	" 1803.	...	1,639	606	...	1 in 1.7.
Miller's Report, pp. 36, 46; Works, p. 89.	" Bellevue Hospital.	...	170	100	...	1 in 2.1.
Med. Repos., vii. 178; Watt's Rept., in Med. and Surg. Regist., p. 359.	" 1805.	{ 75,700, remain- ing 48,774 }	{ 640, Marine Hospital 64— }	{ 292 M. H. 28— }	...	1 in 2.9.
C. Drake, Med. Repos., xxi. 125.	" Bellevue Hospital.	...	704	320	...	1 in 2.3.
Med. Recorder, iii. 207.	" Marine Hospital.	...	149	52	...	1 in 1.4.
Hist. of Proceed. of Board of Health, pp. 243, 247.	" 1819.	120,000	64	28	1 in 2,400	1 in 1.7.
Townsend, p. 198.	" Near Old Slip.	...	70	50	...	1 in 1.93.
	" 1822.	...	63	37	...	1 in 3.
	" 1822.	...	413	244	...	1 in 3.
Rand, Med. Repos., ii. 498.	Boston, 1798.	...	900 (estimated)	300	...	1 in 2.7.
Med. Repos., ii. 334, 392.	" 1802.	250	...	1 in 3.
Brown's Treatise, pp. 25, 109.	" 1819.	42,000	1 in 2.3.
Med. Repos., vi. p. 339.						1 in 1.6
Monson, Additional Facts by Coll. of Phys. of Philadelphia, p. 55.	New Haven, 1794.	...	160	64	...	1 in 3.
Webster's Collection, p. 178.	New London, 1798.	...	246	81	...	1 in 2.05.
Med. Repos., iii. 292, ii. 234, 402.	{ Providence, 1797. " 1800. " 1805.	...	102	45	...	1 in 2.
Wheaton, Med. Repos., x. 335.	Middletown, 1820.	...	83	50	...	1 in 3.
Tully and Miner's Essays, p. 365.	Wilmington, Del., 1802.	...	30	10	...	1 in 2.
Vaughan's Concise Hist., p. 15.		...	15	8	...	1 in 3.
Drysdale, Coxe's Med. Mus., i. 373.		...	197	66	...	1 in 3.
Med. Repos., iv. 205.		1 in 3.
Report of Board of Health.	Baltimore, 1794.	360	1 in 50.5	1 in 2.87.1
Letters and Documents, pp. 176, 177.	" 1800.	60,000	...	1,197	1 in 60	
Revere, Med. Recorder, iii. 222.	" 1819.	...	1,005	350		
Watts, Med. and Surg. Reg., p. 242.						
Reese, p. 40.						

	Alexandria.	{ 6,000, remain- ing 3,000 }	200	{ 1 in 30 entire 1 in 15 remain'g }
Dick, Med. Repos., vii. 390-1.	Norfolk, 1800.	250	
Selden and Whitehead, Med. Repos., iv. 336.	" 1801.	250	
Ibid., vi. 251.	" 1821.	160	
Archer, Med. Recorder, v. 61.						
Ramsay's Hist. South Carolina, ii. 82.						
Simon's Report to Board of Health, p. 7.	Charleston.	1 in 6 est'd.
Dickson's Essays on Pract. of Phys., i. 353.	" Marine Hospital, 1839 } (according to Strobel).	90	20	1 in 4.5.
Strobel's Report on Condition of the Marine Hospital, 1839.	" According to Ramsay.	73	23	1 in 3.15.
Ramsay, Charleston Journ., ii. 638.	" Almshouse, 1849.	53	23	1 in 2.8.
Hayne, Charleston Journ., vi. 341.	" 1852.	42,985	1,000 (est'd)	389	280	1 in 3.5.
Dawson, Charleston Journ., vii. 858-9.	" Roper Hospital.	254	85	1 in 4.58.
Simons, Charleston Journ., viii. 366, 7.	" 1854, Roper Hospital.		92	1 in 2.76.
Wragg, Charleston Journ., x. p. 85.						
Seagrove, Med. Repos., xiii. 139.	St. Mary's, Georgia.	500	132		48	1 in 10.4
Waring, pp. 3, 4.	Savannah, 1820.	Remaining 3,000	666	1 in 5
	Natchez, 1817.	{ 3,000, remain- ing 2,000 }	{ 134, Perlee says 300 }	
Monette, Observations, &c., pp. 63, 102-4.	" 1819.	Rem'g about 1,000	400 (est'd)		180	1 in 2.2.
Monette, Essays, pp. 56, 57, 63, 64, 74, 76.	" 1823.	784	312	1 in 2 est'd.
Perlee, Phil. Med. and Phys. Journ., iii. 6.	" 1825.	150	
Tooley, pp. 17, 8.	" 1829.	87	1 in 3.3.
	" 1837.	300	1 in 2.
	" 1839.	Remaining 850	600 (est'd)	...	235	1 in 2.15.
Monette, Observations, p. 112.	Franklin, Miss., 1839.	250	45		25	1 in 1.8.
Stone, New Orleans Journ., p. 179.	Woodville, Miss., 1844.	800, rem'g 600	595!		60	1 in 9.9.
Lewis, New Orleans Journ., 1848, p. 39.	Mobile, 1839.	...	1,950 (est'd)		650	1 in 3.
Ibid., ii. 290.	" 1842.	...	170		70	1 in 2.4.
Nott, Charleston Journ., iii. 1.	" 1844.	...	169		83	1 in 2.
Ib., New Orleans Journ., x. 583.	" 1847.	...	800		78	1 in 11.
	" 1853.	Remaining 17,000	1,191	1 in 14.26
Marks, Trans. of Med. Assoc. of Ala- bama for 1853, p. 100.	Selma, Alabama, 1853.	{ 3,500, remain- ing 1,000 }	120		32	{ 1 in 109 entire 1 in 31.2 rem'g }

¹ According to Reese, the cases numbered altogether about 1,200, and the deaths about 300; giving a proportion of 1 in 4.

III. UNITED STATES—CONTINUED.

AUTHORITIES.	PLACES.	POPULATION.	NUMBER OF CASES.	MORTALITY.	PROPORTION TO POPULATION.	PROPORTION TO CASES.
	New Orleans, 1817.	24,196	...	600	1 in 40.32	{ 1 in 6 of adult whites, 1 in 10 of all others.
	" 1819.	26,183	...	425	1 in 61.60	
	" 1820.	38,000	
	" 1822.	{ 27,176 (Barton) 44,500 31,706 (B.) }	...	400	1 in 67.92	{ 1 in 1.46. 1 in 1.41. 1 in 1. 1 in 1.54. 1 in 1.59. 1 in 2.02. 1 in 5.2. 1 in 4.8. 1 in 3.41. 1 in 2.23.
	" 1822, Charity Hosp.	...	{ 349 337 ^a	239	...	
	" 1823,	...	1	1	...	1 in 1.46.
	" 1824,	...	167	108	...	1 in 1.41.
	" 1825,	...	{ 94 99 ^a 26 24 ^a	59	...	1 in 1.
	" 1826,	...	372 (NOTE)	49	...	1 in 1.54.
	" 1827,	...	290	5	...	1 in 1.59.
	" 1828,	1 in 2.02.
	" 1829, City.	47,561	...	109	...	1 in 5.2.
	" 1829, Charity Hosp.	...	435	130	1 in 52.84	1 in 4.8.
	" 1830,	...	256	900	...	1 in 3.41.
	" 1831,	...	3	215	...	1 in 2.23.
	" 1832, City.	55,084	...	2	...	1 in 2.02.
	" 1832, Charity Hosp.	...	26	117	...	1 in 2.18.
	" 1833, City.	57,713	...	400	...	1 in 1.5.
	" 1833, Charity Hosp.	...	422	18	1 in 137.71	1 in 1.44.
	" 1834,	...	150	1,000	...	1 in 2.
	" 1835,	...	505	210	1 in 57.71	1 in 1.57.
	" 1836,	...	6	95	...	1 in 1.77.
	" 1837, City.	68,229	...	284	...	1 in 1.2.
	" 1837, Charity Hosp.	...	998	5	...	1 in 2.25.
	" 1838,	...	22	1,300	1 in 52.48	1 in 1.29.
	" 1839, City.	{ 75,000 73,487 (B.) }	In 892 cases	442	...	1 in 13.1.
	" 1839, Charity Hosp.	17	...	1 in 2.4.
	" 1840,	...	1,086	68	1 in 91.85	1 in 1.
			3	Total 800	...	
				452	...	
				3	...	

Report of Med. and Phys. Society, 1821, p. 10.
 New Orleans Med. Journ., i. 103.
 Simond's Statistics of Yellow Fever, in Fenner's Reports, i. 123.
 Report of Board of Health, 1841.
 Report on Epidemics of 1839.
 See, also, Revue Méd., Dec. 1840.
 Thomas, 1st ed., p. 31.
 Ib., 2d ed., pp. 216-17.

NOTE.

CHARITY HOSPITAL.—The statements given in the table are taken principally from the official report of the administration of that hospital, published in the *New Orleans Med. and Surg. Journ.* (i. 103). The late Dr. Drake, in the second volume (p. 197) of his work on the diseases of the Mississippi Valley, has inserted a table compiled by him from data collected from the books of that establishment in 1844. A comparison of these two documents will show several discrepancies of an extraordinary character, a few of which deserve to be noticed. In 1827, the number of cases admitted amounted, in the official report, to 372, and that of deaths to 109. Dr. Drake says, 388 cases, and 265 deaths. In 1830, the numbers, according to the report, were 256 cases, and 117 deaths; according to Dr. Drake, 416 to 158. In 1833, the former document gives us 422 cases and 210 deaths. Dr. Drake found the admissions rise to 887 and the deaths to 449. Again, in 1837, the administration reports 998 cases, with a mortality of 442, while Dr. Drake states the number of cases at 1,194, and that of the deaths at 613.

As with us, the disease in Europe, in various parts of this country and in tropical climates, attains its maximum mortality at diversified periods of the season. Thus, as regards the epidemic of Gibraltar in 1804, the fever reached its culminating point of fatality in the first week of October. In 1813 and 1828, the mortality was again largest in that month. Out of eleven hundred and seventy deaths, reported among the military in the latter year, seven hundred and four occurred from the 29th September to the 2d of November, inclusive.¹ Of twenty-three of the cities of Spain, visited by the fever in 1804, sixteen suffered more in respect to mortality in October.² The same took place at Barcelona in 1821. Of eight thousand five hundred and forty-two interments enumerated in the official report, seventeen hundred and thirty took place in September, five thousand one hundred and forty-three in October, fourteen hundred and thirty-seven in November, and two hundred and thirty-two in December.³ In this country, the disease has occasionally produced a larger mortality in October; as, for example, in Wilmington in 1802,⁴ in Natchez in 1839,⁵ in New Orleans in 1823 and 1846.⁶

More frequently, perhaps, the largest mortality in this country has taken place in September, as illustrated by the epidemics of New York in 1799⁷ and 1822,⁸ of Providence in 1797,⁹ of Baltimore in 1819,¹⁰ of Wilmington in 1798,¹¹ of Charleston in 1807, '17, '19, '24, '27, '38,¹² of Natchez in 1823 and 1825,¹³ of Mobile in 1839,¹⁴ of New Haven in 1794,¹⁵ and by the greater

¹ Mortality of British Army, p. 9.

² Fellowes, p. 478.

³ Estasto Exacto, &c.

⁴ Vaughan, p. 12.

⁵ Monette, Observations, p. 102.

⁶ Barton on Fever of 1833—Table. Fenner, New Orleans Journal for 1847, p. 465.

⁷ Med. Repos., iii. 197.

⁸ A History of the Proceedings of the Board of Health, p. 243, &c.

⁹ Wheaton, Med. Repos., x. 335.

¹⁰ Series of Letters, i. 177.

¹¹ Vaughan, Med. Repos., iii. 371.

¹² Ramsay's History, i. 86; Med. Repos., iv. 233; Dickson, Phil. Med. and Phys. Journ., iii. 258; Simons, p. 14.

¹³ Tooley, p. 17; Monette, Essay, p. 62; Observations, p. 102.

¹⁴ Monette, Obs., p. 118.

¹⁵ Additional Facts, p. 55.

number of the epidemics of New Orleans,¹ 1819, '20, '24, and '33. Of the twenty-three cities in Spain already referred to, two experienced, in 1804, a larger mortality in September. Five suffered most that year in November. Such was also the case at Leghorn in 1804 (Palloni). Finally, the mortality has occasionally been greatest in August, as was the case in Charleston in 1838,² and New Orleans in 1817, 1839,³ and 1853⁴—a circumstance which has never occurred here.

In Europe and various parts of this country, the ratio of mortality to the number attacked differed, as in this city, at different periods of the same epidemic. More frequently it has been greater at the outset, and has gradually lessened as the season advanced. Such was the case in Boston in 1798. Of the twenty or more patients first seized, not one recovered.⁵ The same fatality attended the first cases at New Orleans in 1839.⁶ At Barcelona, according to Rochoux,⁷ the loss at the outset of the epidemic of 1821 amounted, without doubt, to 19–20 of the sick. Towards the middle of the epidemic, it became less considerable, and at the close it was reduced to less than two-thirds. Rochoux further states that the same progressive decrease of the ratio was noticed at Tortosa, Mesquinenza, and in various other cities of Spain. The same observation was made at Leghorn in 1804,⁸ and in Malaga during the same year.⁹

But, although such has usually been the case, it has occasionally happened, that the ratio of mortality, whether at first decreasing or not, has been much greater at the close of the epidemic. In New Orleans, in 1839, the number of deaths, after the fatal outburst of the disease, greatly lessened; but in September, the ratio of mortality sensibly increased. In the hospital, the ratio for July was 1 in 2.3; for August, 1 in 2.51; and for September, 1 in 1.95.¹⁰ A similar observation was made at Opelousas in 1837.¹¹ In New York, in 1822, the disease became less fatal as the season advanced—the cases of recovery gaining fast upon the number of deaths. But in October, and particularly about the middle of it, “just before the termination of the epidemic,” though the number of cases diminished, the disease once more acquired malignancy and became more fatal than it had been at its commencement; the proportion of deaths being to the sick as 3 to 4.¹² In Baltimore, during the epidemic of 1819, after the beginning of October the number of cases and deaths essentially diminished, yet in those who took the disease after this time, it was still more malignant and fatal than at its commencement.¹³ A similar phenomenon was noticed at Natchez in 1837.¹⁴

¹ Barton on Fever of 1833. Report of the Board of Health, for 1841.

² Simons's Report, p. 14.

³ Gros's Report, p. 6; Rev. Méd., Rep. on Fever of 1839, Dec. 1840, p. 324.

⁴ Fenner, Trans. of Med. Assoc., vii. 471.

⁵ Med. Rep., ii. 333.

⁶ Rept., Rev. Méd. for Dec. 1840, p. 324.

⁷ Page 468.

⁸ Dufour, Observateur Méd. de Marseilles, iv. 55.

⁹ Bancroft, p. 470.

¹⁰ Rept., loc. cit., p. 235.

¹¹ New Orleans Journ., 1847, p. 36.

¹² Townsend, p. 197.

¹³ Revere, Med. Recorder, iii. 226.

¹⁴ Monette, Obs., p. 70.

Similar to what occurs in Philadelphia, the ratio of mortality from the yellow fever in other parts of the United States, and elsewhere, varies during the same epidemic in different parts of an infected locality. Berthe, who notices the fact in a particular manner in reference to the epidemic of Seville in 1800, states that the mortality in some districts did not exceed 1 in 18 or 20 of those affected; in others it reached the fourth, the third, or even the half, of the sick.¹ The same result was noticed in New York in 1791² and 1795.³ In the following season, 1796, the ratio varied considerably in different streets—from 1 in 1.5 to 1 in 14.⁴ In the same city, in 1822, this difference of ratio was very striking, the deaths, compared to the sick, being 11—19, 18—27, 11—22, 7—11, 2—2, 1—3, 7—8, 9—16, 20—33, 4—6, 4—7, 4—4, 2—3, 0—1, 8—12, 7—14, 4—10, 2—4, 5—10, 4—11, 1—2, 0—1, 1—2, 2—5, 0—1, 0—2, 1—3, 8—13, 14—19, 4—5, 2—2, 2—6, 1—1, 1—1, 0—1, 34—65, 28—46.⁵ A considerable difference was, in like manner, observed in New London,⁶ and Wilmington (Del.), in 1798;⁷ in Alexandria (Va.) in 1803,⁸ in Boston in 1802,⁹ in Norfolk,¹⁰ in Barbadoes in 1816.¹¹ Blair found that the original site near which the disease first showed itself, in the epidemic of Demerara in 1838, maintained its virulence—the mortality being 24.15 per cent. In other localities, it was 21.41, 21.66, 16.66, 16.17.¹²

CHAPTER XXVI.

PATHOLOGY.

PAINFUL as the avowal may be, it is a fact, the truth of which cannot be denied, that, notwithstanding all that has been written on the subject of the yellow fever, in this and other countries—all the labour that has been bestowed on an investigation of its causes, characters, and anatomical phenomena—little progress has, so far, been made in a knowledge of the pathology of that disease. The physicians of this city and country have not been backward in their efforts to elucidate this intricate and important subject; and to those who are acquainted with the writings of European and tropical authors on this disease, it need not be told that equal efforts have been made

¹ Berthe, p. 163; Bancroft, p. 466; Manifesto que sobre le pasada epidemia, la Ciudad de Seville.

² Addoms, p. 7.

³ Bayley, p. 57.

⁴ Ibid., Letters from the Board of Health, p. 40.

⁵ Townsend, p. 39.

⁶ Med. Repos., ii. 334.

⁷ Vaughan, Med. Repos., iii. 370.

⁸ Dick, Med. Repos., vii. 191.

⁹ New England Journ., viii. 380; North Amer. Rev., x. 396.

¹⁰ Valentin, p. 101.

¹¹ Ralph, Ed. Med.-Chir. Tr., ii. 55, 56.

¹² Page 34.

abroad to attain the object in question. Many opinions have, from the earliest outbreak of the fever among us, been advanced to account for the phenomena observed; and, on reference, it will be found that these opinions correspond more or less closely to those advocated elsewhere, at various periods in the history of the disease. 1. By a large number of physicians, in both hemispheres, the fever, as we have seen, has long been, and continues to be, viewed as differing in degree only from ordinary autumnal remittents, partaking, like the latter, of the bilious element, and depending, as a matter of course, on the same pathological condition of the same organs as are involved in the others; while by other physicians it is held in the light of a distinct and specific form of fever, presenting anatomical characters different from those of other febrile complaints, and depending, as a matter of necessity, on different pathological changes. 2. By some it is assumed that the efficient cause of the disease, whatever be its nature and origin, and the mode of its introduction into the body, affects the entire system. 3. Others contend, on the contrary, that the cause produces its morbid action on certain organs or tissues, which in their turn derange, by sympathy or otherwise, other portions of the economy. 4. By a number of authorities, the disease thus produced is regarded as possessing, in the early stage, all the attributes of a truly inflammatory complaint based on the existence of sanguine inflammation—a condition which terminates, after a longer or shorter time, in one of an opposite kind, marked by phenomena indicative of a state of prostration or atony, and resulting from the destructive operation of the preceding condition. 5. According to some, this state of inflammation is diffused throughout the whole system, in some mysterious manner which need not be inquired into at present, and becomes secondarily localized in organs not necessarily the same. 6. It is the belief of another set that the disease is the result of the primary, not secondary, inflammation of particular organs or tissues, and of the subsequent affection of other parts associated with those originally implicated in the morbid process. 7. By not a few, again, it is regarded as invariably a disease of an asthenic or typhoid character, with or without local affections, the latter being due to an inflammatory process—which some regard as sthenic or asthenic, arterial or venous—or to congestion. 8. By some, the disease is admitted to assume sometimes the inflammatory, at other times the asthenic character, and to depend, therefore, on different occasions, on opposite morbid conditions of the organism. 9. There are not wanting those who refer the main agency in the production of the morbid phenomena to the affection of some portion, or of the whole extent, of the nervous system. 10. A different set locate it exclusively in the solids, excluding the fluids from any participation, except secondarily, in the diseased process; others, on the contrary, look to the blood for the primary link in the chain of morbid phenomena; finally, by some, the yellow fever is held in the light, not of an independent disease, but of a compound of typhus and ordinary autumnal remittent fevers.

Having in another chapter given many reasons for disbelieving the identity of yellow with ordinary bilious remittent fever, and indicated the points of

dissimilarity between them—in respect, especially, to the phenomena and anatomical characters of both—I need not seek among the facts bearing on the pathology of the latter for illustrations of the nature of the former. In the one, the hemorrhagic element is predominant, and the bilious deficient; while in the other precisely the reverse obtains. In the former, we notice, often from the commencement—generally after a very short stage of reaction—a complete cessation of febrile excitement, and a progressive collapse of the powers of life, a frequent absence of cerebral derangement, and a remarkable retention of muscular strength, and other phenomena, which, as a general rule, are foreign to the latter. All these differences, several of which relate to important and characteristic morbid changes, denote undoubtedly a difference in the seat and nature of the pathological lesion, as well as in the characters of the latter; and the explanation which suits one set of symptoms would be inadmissible in relation to the other.

In the present state of our knowledge, it would be a waste of time to examine in detail the opinion of those who regard the yellow fever as a disease of the whole system, in which all the solids and fluids are implicated simultaneously, and remain so from beginning to last. The words are vague, and convey no definite idea to the mind; for to its advocates the question may be asked—Where does the disease, thus assumed to invade the system in its totality, begin, and what is its nature? But the hypothesis is open to the equally strong objection that, if there can exist a disease equally diffused throughout the whole system—a proposition which may be doubted—no illustration of it can be derived from the yellow fever, which, as its phenomena denote, exhibits an integrity of several functions totally incompatible with the idea of any serious derangement of the organs charged with the performance of those functions. So well founded are these objections, that, among writers who have adopted the phraseology in question, as well as among those who discard it, there are some who place the principal morbid agency in the nervous system, which, however widely ramified, constitutes after all but one of the several organic systems of the economy;¹ while by Dr. Rush and others the bloodvessels are held up as “*the seat and throne*” of this and other fevers. As regards the first of the branches of this theory of universal diffusion, it will be sufficient to remark that though the agency of the nervous system, in the receiving of the impression of the efficient cause, and in the conveyance of that impression to various organs and tissues, cannot, in sound physiology, be denied; although, in some cases, the effect of the cause is of so deadly a character as to occasion, by an abstraction of nervous power, symptoms of sudden and great prostration, unattended with reaction, and followed by death; and although the reaction which follows in other cases may be due to what Dr. Wilson (p. 200) denominates an *obstruction* of the nervous power, and cannot occur without the instrumentality of the latter—yet we find no reason in these circumstances for referring the pathology of the yellow fever mainly to a derangement of the nervous system, or for regarding that system as constituting the principal and

¹ Wilson, p. 209; Townsend, p. 179.

primary seat of the disease. Obstruction, as well as abstraction of the nervous power, as manifested by vertigo, faintness, rigor, and failure of the senses, and the like, in the onset, followed by vascular reaction or prostration and congestion, are as frequently noticed, if not more so, in other diseases, in which we should vainly strive to discover a remote analogy with the yellow fever; and yet, if we look to these morbid changes for a pathological explanation of the characteristic phenomena of the latter, no cause can be assigned why we must not attribute to them also the principal agency in the production of other complaints in which they may chance to occur. The circumstance then of the existence of these conditions of the system in yellow fever, common as they are to other and different complaints, will not alone afford a clue to the true pathology of that disease. In the yellow fever, as in other complaints, the instrumentality of the nervous system may justly be regarded as limited to the reception and transmission of morbid impressions, and as the link of sympathetic associations of various organs. In order that the yellow fever may be produced with such phenomena as to assume its characteristic garb, the specific cause must penetrate the organism, and the specific impression must be thence, as it were, transmitted to other portions of the economy, and there give rise to such morbid changes as will occasion those external signs by which the disease is recognized. Until this transmission is effected, we may have a mortal disease, but we cannot be certain that we have the yellow fever; so truly, indeed, that, in cases where the abstraction of nervous power is the most conspicuous, we recognize the disease not by symptoms of the nervous class, but by those which indicate the implication of the capillaries—black vomit, &c.

There are not wanting reasons, indeed, to think that, in the latter, the influence of the nervous system, in giving rise to the characteristic phenomena is more restricted than is generally thought, and that when this system is affected it is so only partially and in a secondary manner through the instrumentality of other parts more early assailed. So far as the nerves of relation are concerned, we have the proof of this in the very frequent integrity of the cerebral functions during the first stage, and in the ordinary integrity of those functions to nearly the last moment of life in the congestive or intense form of the disease, in the absence generally, and until late in the disease, of what are denominated nervous symptoms, and in the circumstances that in cases presenting a contrary aspect, the effect is due often to morbid vascular changes in the brain and its appendages, and, at other times, to sympathetic functional derangements of those organs.

If any portion of the nervous system may be said to exercise, by its diseased modifications, a decided agency, different from that already mentioned, in the production of some of the peculiar phenomena of the fever under consideration, it is the ganglionic, to which some writers attach, as we have seen, much importance.¹ The constipation of the bowels, the restlessness, lassitude, sighing—the pain in the back, limbs, loins, fore-

¹ Gillkrest, ii. 278; Kelly, xiv. 387; Cartwright, ix. 37; Wragg, x. 77.

head, eyes—the gastric irritation, as well as the suspension of the secretory processes—the retardation of the pulse and the diminution of cutaneous heat, the hiccup, &c., afford strong grounds for the belief. But on this subject it may be remarked, that these symptoms do not alone characterize yellow fever; that in all cases in which they occur, an extension of disease has already taken place to the capillaries of various organs, and that they are preceded by a condition of other parts which enables us to explain their occurrence, and the abstraction of nervous power on which they depend; and that their occurrence, as well as that of phenomena resulting from an affection of the nerves of relation, only proves that the nervous system becomes implicated in the link of morbid effects, not that its derangements are to be held up as the foundation upon which our views of the pathology of the disease are exclusively to rest. In a word, we must ascend higher in the scale of morbid action than the affection of the nervous system in order to arrive at the primary morbid agency in the production of the characteristic phenomena of the yellow fever, and seek elsewhere for the seat of the changes which give rise to the manifestation of these.

But although the symptoms of the disease indicate satisfactorily that in the vascular system we are to look for the location of those changes which give rise to the most characteristic symptoms—although we may be justified in viewing, with Dr. Rush, that system as the “seat and throne” of the disease, still, notwithstanding, the admission must be made with some limitation, especially as regards the peculiar morbid changes undergone by the bloodvessels, and the extent to which those changes are carried. By a large number of writers the latter are, as already remarked, referred to inflammation; and to this opinion they are inclined by a review, as they say, of the symptoms, the effect of remedial means, and the result of post-mortem examination. Such has been, and such continues to be, the opinion generally entertained on the subject as regards the yellow fever of tropical climates, of this country, and of Europe;¹ and there is no doubt that, so far as relates to a

¹ O'Halloran, p. 200; *Ib.*, *Fev. of Andalusia*, pp. 75–6; Burnett, pp. 10, 19; Humphreys, in *ib.*, p. 352; Bancroft, pp. 5, 53; Dickinson, pp. 8, 41–2; Wilson, p. 8; Veitch, p. 132; Williams, pp. 20, 37; Catel, p. 13; Tommasini, i. 87; Evans, p. 282; McArthur, p. 351; Dubreuil, viii. 329; Trotter, i. 337, ii. 100; R. Jackson, *Sketch*, i. 60, 64, 331; *Ib.*, *Tr.*, p. 279; Doughty, p. 11; Jolivet, p. 13; Dupont, p. 22; Boyle, p. 287; Barry, in *ib.*, p. 272; Chambolle, *An. de la Méd. Physiol.*, xiii. 202; Le Rivérend, *ib.*, xii. 535, 543; Chervin, *Exam. des Principes*, &c., p. 45; Lefort, p. 575; Rufz, p. 37; E. H. Smith, p. 143; McLean, p. 173; Macmillan, *Edinb. J.*, x. 37; Maher, pp. 841, 894; Monson, p. 186; North (in Pym), p. 33; Belcher, xxiii. 252; Proudfoot, xxvii. 251; Ferrari, *Edinb. J.*, xix. 368; Curtin, *Duncan's Com.*, xix. 236; Palloni, p. 8; Wallace, *Edinb. J.*, xlvi. 279; Vatable, *An. de la Méd. Physiol.*, vii. 309; Jourdain, iv. 548, v. 262; Gilbert, p. 78; Chirac, i. 85; Barton, p. 11; Potter, p. 53; Desperrière, p. 6; Dariste, p. 130; Moseley, p. 430; Waring, p. 46; *Ib.*, *N. A. Med. and Surg. J.*, iii. 1; Hosack, *Practice*, p. 390; *Ib.*, *Essays*, iii. 424; Caldwell (1805), p. 103; *Ib.*, *Essays*, p. 216; Ticknor, iii. 224, iv. 3; Monges, ii. 55, 63; S. Jackson, p. 80; *Ib.*, *Am. J.*, xiv. 43; Grant, p. 37; Drysdale, i. 140; Merrill, ii. 222; ix. 242; Archer, v. 70; Barnwell, p. 373–6; Jameson, *Med. Rec.*, vi. 435; Ffirth, pp. 29, 36; Osgood, p. 22; Nassy, p. 28; Valentin, p. 57; Gros, p. 19; Rochoux (F. A.), p. 336; *Ib.* (F. J.), p. 194;

number of cases, the opinion is correct—the more especially if we regard a decided arterial reaction as indicative of the existence of more or less inflammation. But it is not the less true that, in other cases, there is no evidence of the existence of such a pathological state. The details into which I have entered on the subject of the symptoms must leave no doubt on the mind, that in the more ordinary forms of the disease, the absence of inflammation is placed beyond the possibility of doubt. In some, the pulse, though full, is soft, in others it is feeble and slow, or quick. In many the skin is cool, or little above the natural standard, and we look in vain for the inflammatory condition of important organs. In a word, so far from discovering signs of arterial reaction, we find those of prostration of the powers of life; the vessels, large and small, are in a state of torpor, and instead of exhibiting, after death, those changes which experience has taught us indicate the prior existence of inflammation, we find passive congestion of the capillaries, while the blood, from first to last, is far from being in a state of hyperinosis. Inflammation, therefore, however frequently it may be encountered in the yellow fever, cannot be regarded in the light of a necessary effect of the action of the efficient cause, and as an indispensable element toward the explanation of the pathology of the disease. Or, if inflammation be regarded as the rule, we must admit that the exceptions to that rule are very numerous; and that, in most epidemics, they are much more frequently encountered than the reverse condition of things—the symptoms of arterial reaction being accounted for on very different principles. In a word, local inflammation is not an indispensable element in the constitution of the disease, which may, and does often, exist without it, from beginning to end. It does *not* form part and parcel of the complaint, and when found must be viewed in the light of a complication.

The doctrine under consideration, when applied to the disease generally, is faulty to an almost equal extent whatever be the modification of it we adopt; whether the inflammatory state be thought to pervade, with nearly equal force, the whole system of vessels, and to extend from the heart to the most distant and minute capillaries—the local diseases, when they occur, being viewed as secondary effects; or whether, on the contrary, the local inflammation, as has been stated, is viewed as the primary disease, and the general concurrence of the vascular system in the morbid process as of secondary import.

To the attentive pathologist, the idea that in cases bearing the impress of an inflammatory character we are to recognize the existence of a general inflammation pervading the entire extent of the vascular system, and falling sometimes—but not always—on particular organs, must necessarily appear to be open to well-founded objections. Without enlarging on this subject,

Caisergues, pp. 129, 205; Harris, *Am. J.*, xiv. 46; Girardin, p. 38; Thomas, pp. 59, 88; *Ib.*, 2d ed., p. 129; Davidge, p. 114; Chisholm, i. 105; Towne, p. 25; Watts, p. 259; Rush, iii. 141; Halphen, p. 65; Robert, pp. 12, 316; Kelly, xiv. 377; A. Hosack, p. 26; Shecut, p. 118; Miller, p. 50; *Ib.*, *Med. Repos.*, ii. 414; Caillot, pp. 15, 20, 79, 236; Deveze, pp. 34, 268; Boyd, p. 303.

which has reference to other forms of febrile diseases, as well as to the one more particularly before us, I may state a few of these objections.

1. The same reasoning and facts as are adduced to establish the correctness of that theory in relation to the yellow fever, might be used to prove that in the phlegmasia the local disease is secondary; the general arterial excitement, primary.

2. In many diseases, in which we recognize the existence of primary local morbid changes, the efficient causes which give rise to them, produce their effects much in the way that the efficient cause of yellow fever produces its own effects, whether through the medium of nervous impressions; or, as is the case with specific morbid and other poisons, by means of that power of selection of, or affinity for, special organs and parts which they are known to possess and exercise—a power dwelt upon in reference to the cause of this very disease more than a century ago, by Dr. Warren, of Barbadoes (p. 22), and the reality of which in regard to numberless other agents has been proved beyond dispute by Orfila, Christison, and other toxicologists. If, in the one case, we admit the localization of the inflammation, why shall we deny it in the other?

3. The symptoms of the yellow fever indicate a condition of organs very different from the one which we might expect to be produced by a widespread and equally diffused inflammatory condition—supposing such condition possible; and show, on the contrary, with as much certainty as do those of the phlegmasiæ generally, the existence, from an early period, of local disease. In all cases attended with inflammatory symptoms, we find proofs of the early suffering of particular organs. And were these local disorders the effects of the general excitement or inflammation, we ought to find them proportioned in extent and force to the violence of the latter; and prevailing only in cases characterized by it, whereas they occur as well in instances in which this vascular excitement does not exist at all, or does so to a very limited extent, as in those in which a contrary result obtains. If, therefore, we admit that while in some cases of yellow fever, signs of local inflammation exist, without the concurrent existence of general reaction, by which alone it would be possible to recognize the existence of a diffusion of inflammation over the whole vascular system; if, in many instances, a general vascular reaction manifests itself without being attended by local inflammation, we cannot withhold the conclusion that the latter, when it occurs, is not the effect of this antecedent reaction, but is produced by some other agencies; and that when local inflammation coexists with febrile excitement of the heart and arteries, and especially when this excitement is kept up beyond the usual time, the latter is probably more the effect than the cause of the former.

4. Symptoms, akin to those indicating the existence of inflammation in yellow fever, are known to be produced not only by causes which affect certain organs through the medium of the circulation, but also by various articles applied to the organs themselves, and therein occasioning the inflammatory process. In another chapter I have adduced facts of this kind, and need not do more here than to allude to them. In most such cases,

particular symptoms, characteristic of the disease, black vomit, yellow tint of the skin, &c., have usually been preceded by more or less fever, while the latter itself was undeniably the effect of the local irritation. And although sometimes the absorption of the morbid agent into the circulation has evidently taken place, and may be regarded as the cause of the morbid phenomena; yet, nevertheless, the existence of local inflammation, prior to the development of febrile reaction, and the dependence of the latter on the former, can scarcely be denied or doubted. Here then are symptoms bearing a resemblance to those of yellow fever undeniably produced by the evident impression of a morbid agent on a particular organ, and attended with general symptoms somewhat resembling those of the disease in question. It were strange that the contrary should take place when the efficient cause of the yellow fever is applied to the system, and that, contrary to what has occurred in the former instance, the local disease should be the effect of a general and diffused inflammation.

5. Were such truly the case—were the local subservient to, and produced by, the general inflammation, we should not expect to find the same organs affected in nearly every case. Sometimes the one, at other times another would take on the inflammatory process—the difference depending on a different state of predisposition of the various organs, and a thousand other contingencies. Now, unless we can suppose, in a wide-spreading epidemic, every individual affected with the disease, to possess the same organic predispositions, it will be difficult to explain this uniformity of local affection, but by admitting that the specific cause of the fever has, by virtue of its power of selection, produced its particular and deleterious effects on those organs for which it has an affinity. In this case, the local inflammation is not referable to the general vascular inflammation, but to a condition independent of, and existing anteriorly to the latter; while, like every other local inflammation by which the yellow fever is often complicated, it may, without fear of error, be viewed as adequate to produce or keep up the general excitement of the heart and arteries which supervene.

6. The results of anatomical examinations do not lend support to the idea of the existence of a generally diffused inflamed state of the vascular system. The statement of the existence of inflammation conveys to the mind the idea of certain textural changes, too familiar to be recapitulated here. While these, on the dissection of yellow fever cases, are found in a small number of organs—and these always the same—the other organs are, some generally, others invariably, found destitute of any signs indicative of the prior existence of such a morbid condition.

7. Finally, the mode of treatment which, so far, has proved most appropriate to this disease, lends support to the opinion here advocated; for, in cases in which arterial reaction exists without signs of local inflammation, it would be unsafe to adopt a treatment predicated on the theory of general inflammation; and, when signs of positive inflammation exist, few of the means found serviceable have any other object than that of removing or soothing local complaints, and all are similar to those in use to combat inflammatory diseases of an undeniably local origin.

From all that preceeds, I feel disposed to conclude that, in cases bearing the marks of the inflammatory character, the phenomena which some pathologists have regarded as indicating the presence of a general inflammation, are simply those indicating the existence of febrile reaction, and that this reaction in the yellow, as in other idiopathic fevers, is often due to, or kept up by means of the morbid influence of local irritation. But, whether so or otherwise, the doctrine of the inflammatory nature of yellow fever, when applied, as has been done, to the explanation of the nature of the disease in all its varieties and grades, is erroneous; for if, supposing the idea of the existence of general inflammation to be admissible, we are led to conclude, from a review of the external signs of the disease, that such a morbid state fails to present itself in a large number of instances, it is not less certain that, in many cases, we cannot discover any unequivocal marks of the prior existence of local inflammation, even in organs which, during life, appeared the most implicated, and which were, in consequence, considered as the starting point of characteristic symptoms. The occurrence is not calculated to create astonishment, for it is a fact too well known to need being dwelt upon—receiving support, as it does, from the results in other complaints—that febrile reaction may, and frequently does exist, and is kept up for several days without the presence of any local inflammation, properly so called.

That instances of the kind are less frequently encountered than those in which such inflammations are detected, may, for what I know, be strictly true. Inflammation exists in a large number of ordinary cases of the several grades of the inflammatory class, as well those in which the symptoms are violent as those in which the disease is protracted. Instances occur in which local inflammation is combined with a depressed condition of the system at large—in which, as has been shown by Dr. Lefort (p. 575) and others, the powers of life are, as it were, locked up by the excessive suffering of the organs implicated. Occasionally, from the rapidity of the case, time may not have been allowed for permanent changes in the tissues affected; or again, the inflamed vessels may have been relieved by hemorrhage or other means, and thereby impart, after death, the idea of a condition very different from that which had existed during life. Nevertheless, admitting all this to be true, it is not the less certain that, as regards many cases, the explanations here offered will not apply, and that in them it is impossible to recognize the existence of inflammation; and this, too, not only in cases unattended with general reaction, but in some in which this reaction has been more or less marked—the effect being due in them either to an overwhelming irritation in the nervous coat of particular organs, or, as is more probable, to the peculiar sedative impression produced by the specific cause floating in the blood, on the heart and arteries, and other impressionable parts.

What the nature of the inflammation, when it exists, may be, has been a subject of controversy among writers. From the great turgescence and engorgement of the veins, “extending to their minutest division and first origin,” it has been inferred that the inflammation is venous and not arterial. (*S. Jackson*, pp. 80–81). By others, who take into consideration the broken

or imperfect reaction which occurs in many cases, the disposition to prostration which manifests itself in most instances, and the dark colour of the affected tissues often noticed after death, it has been argued that the inflammation is of an asthenic character.¹ But it would appear more consonant with the principles of sound pathology to regard the inflammation in this and other diseases as of an active kind, and occupying the same order of vessels, but modified in the former by a greater disposition to capillary or venous congestion, a deficient tone in the vessels, and an altered condition of the blood circulating in them, combined, in some forms of the disease, with an impaired power of vitality of the system at large. At the same time, it is difficult to withhold the opinion, that the inflammatory process is modified not only by the circumstances mentioned, but also, and more effectually, by the specific nature of the cause to which the disease owes its origin. The doctrine of specific inflammation receives ample support from the observation of clinical phenomena; and it has long been remarked, that one of the greatest errors of the schools of Rush and Broussais, was their having advocated the unity of diseased action, and confounded together all irritations of particular tissues—viewing them as differing from each other only in degree, and in the nature of the parts affected. By so doing, they were led to regard as affections of the same kind diseases which, though situated in the same parts, differ more or less in their symptoms and causes, as well as in the treatment required for their cure. The inflammation of the skin which gives rise to erysipelas, differs from that constituting a common ringworm, and that produced by Spanish flies; and yet they are located in the same tissue. The gastro-enteric irritation produced by the ingestion of certain shell-fish, by mushrooms, corrosive sublimate, arsenic, by marsh miasmata, and cold, are certainly not alike, though located in the same part of the economy. Now, when, with these facts before us, we find that the inflammation of the yellow fever is produced by a cause different from that of other diseases, that it gives rise to symptoms of a peculiar character, and that it generally terminates in a peculiar way, we may be pardoned for regarding it as of a distinct or specific kind.

As to the organs or tissues which, in the yellow fever, are most frequently inflamed, and by their morbid derangement give rise to symptoms which impart special features to the disease, thereby affording us a clue to the elucidation of its pathology, we can find little difficulty in tracing them to the abdominal cavity. The inflammation of the lungs and kidneys are too seldom encountered to be of great service in establishing the pathology of the disease, and need not, therefore, occupy us here; while the functions of those organs, when deranged, are so, in the greater number of cases, from other and different causes. The same may be said of the affection of the brain and its membranes. It is true that, by some writers (*Hastings*, p. 41), the disease has been roundly asserted to consist of little more than inflamma-

¹ Gilbert, p. 79; Shecut, pp. 116, 118; Caisergues, p. 129; Chisholm, i. 105; Dubreuil, viii. 317; Cassan, v. 96; Gillespie, p. 63; Dupont, p. 23; Boyle, p. 287.

tion of those parts. It is equally true, also, that by others—somewhat less exclusive in their views—this morbid state, though sometimes found wanting, is regarded as sufficiently often associated with that of other important organs to impart a special pathological element to the fever, and justify its being regarded as forming part and parcel of it, and that it assists us in discovering the name by which it should be designated.¹ But, when we bear in mind what has already been said respecting the frequent or usual integrity of the intellectual functions in the early stages of the disease—their restoration at a subsequent period in cases in which they had been early disturbed—the frequent continuance of their sound condition to the last moment of life in some of the worst forms, as well as the results of post-mortem examinations, which reveal an absence of all signs of inflammatory change, even in cases attended during life with derangement of the intellectual functions, we cannot err in rejecting such views of the pathology of the fever under consideration, and in concluding that inflammation of the parts in question should, when it occurs, be regarded in the light of a complication, which may or may not exist without affecting the question or occupying us at this moment. We must conclude, in like manner, that symptoms usually regarded as indicative of cerebral or meningeal inflammation, may, in the yellow fever, as they do in some other forms of disease, be due to other causes; either to a functional disorder depending on sympathetic connection with other suffering parts, or to the presence in the vessels of the brain of foreign substances—urea, or the elements of the bile—which, in consequence of the disordered condition of their eliminatory organs, continue in, and tend to contaminate, the blood. Neither can we place the seat of the primary inflammation in the ganglionic centres and nerves, and regard that inflammation as affording a clue to the pathology of the disease; inasmuch as its existence, if real, is only occasionally so, and also because it exists in other complaints characterized by symptoms very different from those of yellow fever.

Of the abdominal organs, the liver has, by some, been regarded as principally implicated in the disease, either alone or in connection with other parts. Thus, by Tommasini and others,² the yellow fever has been regarded as a gastro-hepatitis; but, neither the symptoms during life, nor the result of examinations after death, lend support to that opinion. With few exceptions, we find no signs of increased bilious secretion, no pain in the hepatic region; the secretion of bile, indeed, is diminished. The day is past when the matter of the black vomit was regarded as the product of an inflamed or deranged liver. Except in some cases, the discoloration of the skin is far from being exclusively the result of an hepatic cause, and when so, it is due to a cessation of bilious secretion—the result of sedation, or fatty degeneration of the organ, and not of vascular erethism. The changes found on dissection are very different from those occasioned by inflammation, exhibit-

¹ Hence the name of Meningo-Cephalo-Gastritis, given to it by Maher, p. 841; Chambolle, p. 202; Catel, p. 13.

² Tommasini, Sulla Feb. Gialla, i. 87, § 49; Robert, Guide Sanitaire, p. 12, 282.

ing, generally, an anemic, not an engorged state of the liver, and an accumulation of oil in the hepatic cells; while the treatment called for is different from that suited to ordinary hepatitis.

From all which precedes we may conclude that, however true it may be that in some cases, particularly in the commencement, the liver is in a state of irritation, as indicated by increased secretion, or even positively inflamed, such morbid conditions, which are as often seen in other complaints, are too frequently absent in the yellow fever to be regarded otherwise than as accidental complications. They produce, during the course of the complaint, no symptoms required to identify the latter and insure a correct diagnosis, while they leave after death no textural lesions approximating to those which experience has taught us to expect to find in autopsies of true but simple yellow fever cases.

It is true that the liver is found altered in colour and consistence, and containing a greater or less quantity of oil; but these changes are rather the effects of the disease than the disease itself. They are occasionally found in complaints bearing no analogy to the yellow fever, arising from different causes, and characterized by widely different phenomena. They result from a pathological condition the reverse of inflammation and irritation. Wurz and Paget regard fatty degeneration in general not so much in the light of a new deposit as of an unrepaired tissue falling into a state of atrophy and transformation. Whether this be so, or the fat be a new deposit, there is little doubt that the degeneration results from a deranged nutrition; that it is in no way connected with a state of inflammation or irritation of the organ, and is encountered in complaints in which neither of these morbid states exist, but where the process of nutrition is greatly impaired.

When from the liver we pass to the gastro-enteritic organs, and to their mucous lining more particularly, we find ample proof of the necessity of looking there for the most common location of the inflammation. The symptoms during life point, from an early period of the attack, to the stomach as the organ most materially affected—as “the chief seat or throne of the furious conqueror.” One of the main indications in the treatment of the disease is to correct and remove the morbid state of that organ; on examination after death, we discover, in a number of cases, evidences of inflammation, while in not a few others, in which such marks are not visible, we may, for reasons already assigned, presume that the morbid state in question existed, but left no traces behind. So glaring, indeed, is the proof of gastric irritation in the yellow fever—so frequent the signs of inflammation of that organ, that of the pathologists who have regarded the disease as one of inflammatory character many—and of those who view it in a different light some—have considered it as a variety of gastro-enteritis; while others avow that those organs become more frequently than any other implicated secondarily in the inflammatory process, and that this inflammation, by giving rise to various characteristic phenomena of the disease, becomes part and parcel of the latter.¹

¹ Gilbert, p. 79; Barton (1833), p. 11; Nassy, p. 28; Caldwell (1805), p. 99; *Ib.*, Essays (1826), p. 153; Caisergues, pp. 129, 205; Girardin, p. 38; Rochoux, p. 375; *Ib.*,

Nevertheless, though it is impossible to shut our eyes to the frequency of such a morbid state of the organs in question, I scarcely need remark, after what has already been said, that gastro-enteritic inflammation is not an essential element in the pathology of the yellow fever. Gastro-enteritic inflammation does not exist in the early stage of the disease, and is even wanting in the latter stages of many cases. Yellow fever, therefore, cannot with propriety be said to consist of gastro-enteritis only. We must, in cases where such a morbid state exists, go beyond it to arrive at the true pathology of the case. We must reach to some morbid condition, which exists in all cases, and without which the disease cannot be diagnosed. But I repeat, true as it may be that, in many cases, the absence of gastro-enteritis may be easily made out, it would be wrong to argue, from the correctness of the explanation in them, that inflammation exists in no instance, that what many pathologists have regarded as due to the latter morbid state should always be referred to simple nervous irritation; and that the yellow fever is from the outset, and continues throughout, in a great measure at least, free from vascular complications.¹ Important as is the part played by the nervous system in the production of the phenomena of the yellow fever, signs of inflammation during life, and the evidences of the effects of that morbid condition discovered after death, are too frequently found and too well marked to be ignored; and however matters may have stood at the outset, the disease, in most cases, is not long in affecting the capillary vessels, and therein occasioning sometimes inflammation, at others congestion, and generally hemorrhage. Of the existence of the former, enough has been said, and it would be useless to dwell on the tendency to hemorrhage displayed by the disease. As regards the congestive state of the vessels, which, like inflammation, generally precedes the occurrence of hemorrhage, it manifests itself often, and imparts a peculiar character to the cases in which it is observed. So frequently, indeed, is it encountered, so distinctly marked are the signs by which it is distinguished, and so difficult is it sometimes to draw the line of demarcation between that state and inflammation, that there are not wanting those who are disposed to deny the existence of the latter as a natural element of yellow fever, and to lay most of what has been said on the subject to the score of congestion, limiting the existence of the other to those cases in which emetics and drastics have been injudiciously used.² Avoiding all ultraism in this matter, it will be safe to say that, though not existing always to the entire exclusion of inflammation, congestion of the capillaries and larger vessels is of frequent occurrence in the yellow fever; that it often complicates the other

F. J., p. 194; Robert, p. 286; Waring, p. 46; *Ib.*, N. Am. Med. and Surg. Journ., iii. 1; Miller, p. 50; Dubreuil, viii. 329; Caillot, pp. 79, 100, 236; Catel, p. 13; Evans, pp. 260, 282; Barry, p. 272; Chervin, *Examen.*, &c., p. 45; Chambolle, xiii. 202; Le Rivérend, *Ann. de la Méd. Phys.*, xii. 535; Lefort, p. 575; Physick, v. 131; Monges, ii. 63; Harris, *Am. Journ. Med. Sci.*, xiv. 46; Ferrand, *Ann. de la Méd. Phys.*, vii. 299; Chervin, *ib.*, p. 298; Jourdain, v. 259-262; Maher, p. 841; Dickson, *Am. Journ. Med. Sci.*, i. 270.

¹ Chabert, p. 32; Bally, p. 384; Townsend, p. 198.

² Wurdeman, *Am. Journ. Med. Sci.*, N. S., ix. 52; Watts, p. 257.

morbid state mentioned, and as often exists alone. But, after all, it is only an effect, not a cause of the disease, which may, and does, frequently exist without it. To this it may be added that, in many cases, the gastric suffering is evidently due to some form of nervous irritation or deranged condition of the mucous membrane, which is distinct from true inflammation, and leaves no traces after death; or is characterized by a merely congestive state of the capillary vessels, having, like the inflammation before mentioned, a hemorrhagic tendency.

Nor, on the other hand, is it necessary, after all that has been said, to enter at large into an examination of the opinion of those who regard the yellow fever as exclusively and under all circumstances a disease of an adynamic or typhoid character, commencing in debility, displaying throughout its progress the most positive debility, and terminating by the direct destruction of vitality. The number and professional standing of the advocates of this theory are certainly respectable,¹ and their opinions on all subjects connected with the yellow fever are entitled to our serious consideration. But, on the point before us, we can see no just reason to unite with them in regarding the disease as invariably of a typhoid character, and, as it were, a modified form of typhus fever. The symptoms enumerated; the phenomena noticed after death; and, as we shall see, the mode of treatment found successful, as well as the florid condition of the blood, leave no doubt that in some cases the disease is characterized, in the early stage, by a greater or less degree of inflammation; that frequently this condition is evinced by general vascular reaction; while in other instances, prostration of the system at large is accompanied with a condition of organs scarcely compatible with the theory in question. Such facts prohibit us from doing more than admitting that, in some of its forms, the disease assumes a typhoid or congestive character; not that it is invariably, in all cases and under all circumstances, of the nature contended for. Those who uphold this view have, to say the least, given an erroneous explanation of facts which have presented themselves to their observations.² In some instances, they have generalized from a limited number of cases, considering explanations suited to the few instances they had in view, as applicable to all other cases, whether occurring in the same or in different places or seasons. In not a few instances, they have held certain symptoms described, and morbid conditions discovered after death, as indicative of a typhoid state and passive

¹ Daniel, p. 77; Hillary, p. 153; Warren, p. 31; Bayley, p. 113; Berthe, p. 92; Wright, vii. 6, 7; Blin, pp. 6, 7; Imray, liii. 91, 92; Kéraudren, p. 5; Rayer, p. 59; Gillkrest, p. 280; Chisholm, i. 318; J. Clark, p. 21; Bruce, p. 277; Stevens, p. 193; Desportes, i. 191; Lefoulon, Introduction; Holliday, p. 11; Guyon, pp. 44, 45; Nasmyth, in Lind on Seamen; Lefoulon, xi. 103; Bally, p. 385; Pugnet, p. 361; Savarésy, pp. 261, 301; Dalmas, pp. 13, 237; François, p. 12; Tully, p. 304; Powell, pp. 2-5; Moultrie, pp. 17; Bryson, pp. 77, 251; Rouppe, p. 406, Engl. ed.; Blanc, ii. 113; Mabit, p. 9; Pym, pp. 11, 239; Pariset, p. 578; Levacher, p. 79; Blair, p. 104; Fontana, p. 72; Fellowes, p. 308; Lefuerte, in Fellowes, p. 317; Madrid, pt. ii. pp. 3, 13; Lallemant, p. 127.

² Daniel, *Fev. of Savannah*, p. 90, &c; Tully and Minor, *Essays*, pp. 303-307.

capillary congestion, which other pathologists would regard in a very different light. Sometimes, again, they have held up some of the cases on which their theory is predicated as alone entitled to be considered as true yellow fever, and as distinct, in every respect, from others; while a different set of physicians have more properly considered those cases as constituting only a separate form or variety of the same disease—produced by the same causes, presenting the same characteristic symptoms, occurring often at the same time and in the same places—but modified by a variety of influences, telluric, atmospherical, and personal, which have often been enumerated.

By some physicians of a former generation (Blane, Lempriere, Dickson, and Chisholm), the form of fever under consideration—the true and genuine yellow fever—was regarded as a hybrid disease, a compound of the typhus of Europe grafted on the yellow fever of tropical regions. Though he refused at first his belief to this compound character, Chisholm admitted it, openly and decidedly, at a subsequent period. In his history of the epidemic of Grenada, after stating that he had, in his first edition (p. 146), said that by some writers the fever of Grenada was thought to be the yellow fever of the West Indies engrafted on the European jail fever, he adds: “But it does not follow that I embraced the same way of thinking.” Twenty-one years after, he appears to have changed his views on the subject, for in the opening paragraph of his chapter on the yellow fever, which, as is well known, he denominated “malignant pestilential fever,” contained in the *Manual of the Climate and Diseases of Tropical Countries* (p. 167), the author says: “It must be kept in mind that this, the most tremendous of all the tropical diseases, wherever it appears, is the typhus of Europe grafted on the yellow remittent fever of the torrid zone, or of countries whose climate, during part of summer and autumn, possesses the temperature of the torrid zone.” “A fever originally typhus, the infection of which there are solid grounds for believing, was received, prior to their departure from England, by the persons and clothes and bedding of many of the adventurers who were embarked, and who perished in the attempt to colonize the island of Bulam, on the coast of Africa, was introduced into the island of Grenada, in February, 1793, by the ship Hankey, one of the ships employed in the attempt. This fever possessed the peculiarity I have mentioned, of having the symptoms of yellow remittent fever superinduced on those of typhus.” “This singularly mixed character of fever was unknown to me and to all the medical gentlemen of the island with whom I communicated on the subject.”¹

On the subject of the hybridity of the pathological nature of the yellow fever, it need only be remarked that although disposed, as the contents of a preceding chapter will show, to admit the occurrence, in a greater or less number of instances, of complications akin to those referred to by Chisholm, I cannot suppose that any one, at the present day, will feel disposed to admit that the yellow fever can in any way be a compound of two diseases, one of which has not the remotest analogy to it. Besides, let it be

¹ Page 168. This work was published in 1822.

remarked that all the symptoms and anatomical lesions encountered in the Grenada fever, or on other occasions, by the authors referred to, are noticed in the yellow fever of this country and elsewhere, under circumstances which forbid the idea of such an origin. They indicate only a particular form of the one fever—which is noticed everywhere else within the yellow fever zone, not a distinct disease formed of the elements of two others—a form which occupies the ground exclusively in some seasons, and at other times shows itself in only a comparatively few cases amid a large number of a different kind, and consequently does not differ materially from these in a pathological point of view.

From a consideration of these circumstances, which it would be unnecessary to elucidate minutely here; from a review of all the phenomena of the disease; of the nature and mode of introduction of the agent to which it owes its origin; of the condition of both the solids and fluids, and the result of post-mortem examinations, we may draw the following conclusion as respects the pathology of the yellow fever. The cause of this complaint, however it may have originated, is a special poison, which, having penetrated the system and entered the bloodvessels in a manner well known, is carried everywhere through the instrumentality of the blood. It produces in that fluid changes, to which attention was called in a former chapter, and others of a more intimate kind, which escape the cognizance of our senses; and by means of the polluted blood, occasions important morbid changes in some of the abdominal organs. It produces, besides, a morbid impression on the nervous centres; and these, as well as the former, in their turn, induce, by virtue of the numerous sympathies which bind together the various components of the system, diseased modifications in various organs and tissues, and thereby give rise to phenomena of a more or less peculiar kind, or essential to the manifestation of the fever, and varying somewhat in number and violence in different individuals and under different circumstances. The morbid actions thus produced may be combined with opposite conditions of the vital forces, and may or may not be accompanied with inflammation of the parts primarily or secondarily affected; these and the other differences alluded to depending on age, habits of body, and numerous other circumstances connected with the individual; on the degree of concentration and violence of the efficient cause; on the peculiar character of the epidemic constitution of the atmosphere; on the nature of the localities, and numerous other modifying agencies. Hence, in some cases—under some circumstances, in the majority, if not in all—the morbid action of the poison is such as to destroy at once the powers of life; or, in less rapidly fatal instances, to greatly diminish them in all the parts to which it penetrates, and by that means, as well as by the diminution of fibrin or other changes occasioned in the blood, to impede or annul the force of reaction inherent in the system—impairing or destroying the vitality of the tissues and organs, checking the secretions, modifying and vitiating the power of nutrition, lowering the tonic force and vital energy of the bloodvessels, promoting the formation of capillary and venous congestions, facilitating the escape of blood from or in the substance of the tissues, &c.

The disease, in such cases, presents itself clothed in the garb appertaining to the asthenic and congestive forms described in a former chapter, in which there is, strictly speaking, no fever, but in which the identity of the complaint is made out by peculiar symptoms which display themselves from the outset to the close of the attack. In other instances—which, in certain seasons and in certain places constitute the majority—the impression of the poison is soon followed by reaction which becomes more or less marked—continues for a longer or shorter time, with the usual symptoms of such a pathological condition—vascular excitement and often florid blood—and is succeeded either by recovery, or, after from two to four days, by a state of collapse and disorganization akin to that which in the other forms occurred from the outset.

The yellow fever, in such cases, assumes the inflammatory character, and, like the asthenic or congestive forms, is susceptible of being classed into several varieties. In many instances, especially of the inflammatory, though sometimes of the asthenic form, the disease shows itself in combination with local inflammation of various organs—particularly of the gastro-intestinal mucous tissue. In other instances, on some occasions in the larger number, those local inflammations are replaced by a greater or less degree of congestion of the capillaries of the above and other tissues, and of the larger vessels. In others, again, both conditions are combined, and in all severe and fatal cases, the capillaries, at an advanced period, lose their tone and become the seat of passive hemorrhages. In a word, the modifying agencies to which I have alluded, may so interfere with the action of the poison as to give to the fever which it produces a character altogether different from that which it usually presents, just as a peculiar habit or a peculiar idiosyncrasy will sometimes enable the individual to resist the reception, or at least to throw off or eliminate the poison when received, and escape its deleterious action altogether.¹

That the disease is a general one we may infer from a circumstance to which attention has sometimes been called by pathologists. Jaundice, as Mr. Maher remarks, constitutes a phenomenon of too prominent a kind to allow it to have been forgotten in the description of the disease. So far from this, it has been amply dwelt upon everywhere and by every writer, and has even given a name to the disease under consideration. Yet this symptom often fails to present itself. The same may be said of the black vomit, so that the yellow fever may really exist without being characterized by either of those symptoms. This proves to my mind, as Mr. Maher states, that the disease to which these two names have been given is not a local affection as these nomenclatures would lead us to infer. If the morbid changes were confined to the stomach and to the liver, the effects produced should always be the same, and the black vomit, as well as the jaundice, should never be absent. If, on the contrary, the lesion which constitutes the yellow fever is generalized and distributed at the same time to one or more apparatuses, we

¹ Wallace, Edinb. Journ., xlv. 277.

may readily understand that some of its phenomena of outward expression may not be fully developed without its being possible on that account to doubt the identity of the disease.¹ That the yellow fever does, like other great epidemics, give rise in the course of its progress to morbid changes in particular organs no man of sense and of common observation will deny; such effects are to be expected from all poisons which penetrate into the blood, or act in any way on the system. Sometimes in this disease there is gastritis; the liver is generally changed in a remarkable degree; the kidneys are found altered in colour and texture; the lungs show dark patches; the brain is congested and bloody tumours form; the secretions are checked; the texture of the skin is so changed that I have seen the cuticle, by attempting to cleanse the face with a towel wiped off and the skin left as raw as a blistered surface, and this, too, twelve hours before death. Who would say that smallpox and plague were localized because they affect the skin or glands, and so with the gastritis of yellow fever; nor is there any locality which can be assigned to inflammation which will account for the tendency to rapid collapse, the state of the blood, the depressing effects often seen from depletion, the early demand for stimulants.²

The importance here attributed to the agency of the blood in the production of the yellow fever, the belief expressed of this fluid being the recipient, and transmitter to the tissues and organs of the special poison to which the disease owes its origin; of its being itself tainted by that poison, and becoming gradually in severe and fatal cases perfectly disorganized; the opinion of its producing by that means a more or less profound derangement in the organism, and thereby occasioning the symptoms characterizing the disease; in other words, the theory of the fever being the result of a morbid intoxication—of the introduction into the blood of a morbid poison—whether contagious or not matters little in this place—has long been, and continues to be upheld, in a more or less modified manner, in this and other countries. Everywhere it is advocated by writers³ of more or less note, in works to which reference has often been made, and is, as I need scarcely remark, an offshoot of the old and much and unjustly abused doctrine of the humoral pathology. That it will shock the theoretical notions of exclusive solidists, there can be little doubt; but although every enlightened physician in this age of medical improvement, must feel disposed to discard the fanciful speculations and false assertions of the exclusive humoralists of former times, experience and a more careful mode of investigation have opened our eyes to the impropriety of going to the other extreme, and shown the necessity of admitting that, in one class at least, of disease, the pyrexia, the blood plays a more important part than is commonly supposed. As regards the

¹ N. O. Journ., x. 467.

² Nott, Am. Journ., N. S., ix. 288.

³ Townsend, pp. 195–199; Moultrie, p. 19; Harrison, v. 580; Williams, on Morbid Poisons, ii. 466; Hillary, p. 153; Imray, liii. 91; Warren, p. 24; J. Hunter, p. 155; Th. Clark, p. 5; Rochoux, pp. 146, 541; Stevens, pp. 163, 188; N. O., 1839, p. 336; Copland, iii. 200; Nott, Am. Journ., N. S., ix. 288; Bertulus, p. 17; Lefoulon, p. 56; Levacher, pp. 78, 79, 80.

yellow fever in particular, the pathological view here advocated recommends itself not only from the respectability of the authorities by whom it is upheld, but from the nature of the facts and considerations upon which it is founded. Difficult, indeed, would it be, in the present state of knowledge, to venture on any other conclusion, when we call to mind the toxicological nature of the efficient cause of the disease and the mode of its introduction into the system—the morbid changes which take place in the blood, even at the outset of the attack, and which end more or less rapidly in a diminution of fibrin, a deficiency of adhesion and incipient putrefaction, &c. ; and the greatly altered condition of that fluid as found after death in the heart and large vessels ; the production of morbid effects akin to those of the disease, by the artificial introduction of putrid or other substances into the blood. To this let us add, the communication of the disease to the fœtus in utero, the tendency to capillary congestion, and the frequency of passive hemorrhage, external and internal, which experience teaches us is always the consequence of an attenuated blood, however produced ; the discoloration of the skin ; the rapid putrefaction of the body after death ; and the inconstancy of pathological changes in the tissues capable of accounting for the disease—especially those of an inflammatory character. These facts, as well as the circumstance that the disease often ends fatally without leaving traces of important changes in tissues and organs which from the phenomena noticed might have been supposed to be affected, all indicate the operation of a morbid poison acting through the medium of the blood ; while we can discover no fact to justify the belief, that the first link in the chain of morbid effects may be traced to a disturbance in the function of innervation, or to the diseased condition of particular organs.

CHAPTER XXVII.

DIAGNOSIS.

THE discrimination between the yellow fever and other febrile complaints, is a subject which, under ordinary circumstances, can prove of little difficulty to a physician familiar with the pathognomonic phenomena of the disease, and of those with which it is contrasted. Doubtless, if we take a survey of the symptoms of the yellow fever, we shall soon be convinced of our inability to find one which, when viewed by itself, and unconnected with others, or without reference to the degree of its frequency, can, strictly speaking, be looked upon as really pathognomonic of the disease—in other words, as imparting to it an idiopathic or special character, and as contradistinguishing it from all other forms of febrile complaints. Each is, at times, absent in cases of undoubted character ; and each will be found to occur in other diseases but remotely connected with the former. Thus, the yellow suffusion of the skin

is not an unfrequent attendant on a variety of morbid states very different from the yellow fever, and, as we have seen, sometimes fails to show itself in the latter. The black vomit, as has been also shown, may be absent in cases as to the character of which there cannot be a doubt—sometimes in fatal, and, with exceptions, always in cases of recovery; while it has been observed in diseases which, on no account, could be mistaken for the disease in question. The same may be said of the burning heat and irritability of the stomach, of the state of the eyes and tongue, of the condition of the intellectual faculties, and of the nervous system generally, of the pulse, skin, countenance, and of the type which the fever assumes; while the symptoms by which an attack is ushered in, frequently bear so close a resemblance to those noticed in other fevers as to offer but slender means of arriving at a correct diagnosis. But, however true all this may be, it is no less certain that when viewed in connection with each other—when found associated together, or when the greater number of them are present in the same case—especially when this assemblage is found to hold in a large number of individuals affected, the result is different. Under those circumstances, the peculiar jaundice described, varying from the bright yellow to a dark mahogany or livid hue—the emission from the stomach of the dark coffee-ground matter, so well known under the name of black vomit—the injected, brilliant, transparent, fiery, and glassy eye—the thin, slimy, white or moist, thick and dirty yellow fur, and clean, red edge and tip of the tongue—the super-orbital pain—the rachialgia—the single febrile paroxysm, and its sudden cessation at the end of from some forty to seventy-two hours, more or less—the absence, from that period, of all fever—the progressive increase in the slowness and depression of the pulse—the gradual loss of cutaneous heat—all these may be viewed, in their *ensemble*, as typical of the disease, and as its characteristic and pathognomonic phenomena. When they all occur together, or when only one or two fail to do so, the physician may be assured he has to deal with the yellow fever; and the certainty is enhanced when the case in which they are observed presents itself at a period of the year, and under circumstances favourable to the development of the disease. On the other hand, when they are all, or for the most part, absent—the black vomit and jaundice particularly—we may, in the majority of cases, safely conclude that the disease is of a different kind, even when circumstances are favourable to the development of the yellow fever cause.

The yellow fever presents itself, in some instances, in so mild a garb—more frequently during some epidemics than others—the symptoms are, under those circumstances, apparently of so trifling a nature, and the disease bears, at first sight, so great a resemblance to a common ephemeral fever, that no censure can be cast on the physician who, when meeting with such cases, experiences difficulty in establishing his diagnosis, or even when he gives a decided opinion against their yellow fever nature. The difficulty, in these cases, is especially great when attacks of the kind are few in number, and scattered over a large surface; or when they occur in the early part of an epidemic, and are not contemporaneous with others of a more violent or

decided character; for the attention of the medical attendant is not specially directed to a minute examination of all the phenomena presented, or, deprived of objects of comparison, his suspicions are not aroused by observing certain links by which the cases before him are connected with those existing at the time, and on the nature of which there can exist no doubt. Nor is the difficulty lessened by the fact that, in some of those instances, yellowness of the skin supervenes, and that in a few the disease, after presenting, at first, the character mentioned, assumes a more malignant form, and the patient, besides being jaundiced, throws up from his stomach a fluid of a dark brown colour; for such symptoms are observed as well in other febrile diseases, as in true yellow fever, though of course not so frequently.

Difficulty may arise in the early stages—those of the outset and of tumult—and great stress has been laid on this by the opponents of the independent nature of the disease from the circumstance that, at this early period, it is often impossible to foretell whether the case examined will turn out to be one of yellow fever or of some other disease, and that we must wait the appearance of the graver symptoms—as black vomit—before establishing the diagnosis of the case. The difficulty at that early period is acknowledged, but proves nothing relative to the non-independence and specific nature of the complaint, or to the impossibility of distinguishing it from others. The same difficulty is often felt, even by experienced physicians, in the case of diseases, the nature and causes of which differ essentially, and which approximate only in being attended by symptoms of constitutional excitement. On this subject I need hardly insist. It is, or certainly ought to be, admitted by all who have the least smattering of practical medicine. If this be true in reference to such diseases, the embarrassment may well be admitted to occur much more frequently when the complaints to be diagnosed are of the same family, though of different species; for all know that in such diseases the full development of the phenomena which serve to characterize the case, is usually preceded by febrile symptoms very similar in all, and well calculated, on that account, to create difficulty in respect to the diagnosis. If we take, for example, the incubative symptoms in continued fever, we shall find that the description of them may apply to many of the acute blood or zymotic diseases.

The following catalogue of symptoms I borrow, like Dr. Simons, from Dr. Watson's matchless book on the practice of medicine: "The expression of the patient's countenance alters; he becomes pale, languid, and abstracted; those about him observe that he is looking very ill. He is feeble, and easily tired; reluctant to make any exertion of mind or body; listless, and often apprehensive of some impending evil; he loses his appetite; his tongue becomes white, and inclined to tremble; the bowels are irregular, often confined, sometimes affected with diarrhoea; his senses lose their natural delicacy. He has uneasiness or wandering pains in various parts of the body, and occasionally there is some giddiness; drowsiness, perhaps, during the day, and unsound, unrefreshing sleep at night. In a word, the patient droops. The regular onset of the fever is very frequently, indeed, marked by a shivering fit; another common phenomenon, at the period of the inva-

sion, is severe headache. But you will also perceive, even when there have been no premonitory circumstances, that symptoms arise, even thus early, which belong to the nervous system, and denote some disturbance and alteration in the functions of sensation, thought, and voluntary motion. They are comprised under the general phrase, 'febrile oppression,' and they are different from what we notice when pyrexia or feverishness supervenes upon inflammation. The muscular power is sensibly enfeebled. Sometimes the patient will struggle against this, but in a few hours, or in a day or two at farthest, he takes to his bed."

To these symptoms, which, as Dr. Simons remarks, constitute equally a picture of most of the diseases of the class, there succeeds a stage of reaction, in which fever runs more or less high. The skin is hot and dry, sometimes moist; the pulse is excited, thirst is developed, and so on of other symptoms familiar to all physicians; and it is only after these have continued a longer or shorter time that the characteristic phenomena manifest themselves, and the true nature of the case is satisfactorily made out. Exceptional cases, doubtless, are found, in which, from peculiar signs, aided by various concomitant circumstances, the diagnosis may be established earlier. But, in general, it is not so, and every prudent physician finds it better to avoid precipitancy in the expression of his opinion. With Dr. Nott, therefore, we must all concur, when he remarks: "If a physician were called in the forming stage of a number of cases of the plague, smallpox, yellow fever, some forms of typhus, and other diseases arising from morbid poisons, as well as certain vegetable poisons, he would be much at a loss how to distinguish them for two or three days; and in some of those in which the characteristic signs are never developed, as smallpox without eruption, &c., a diagnosis never could be made. It should not be wondered at, then, that difficulty of diagnosis should sometimes occur between bilious and yellow fever, which belong to the same family, the same season, and (often) the same locality."¹

The writer last referred to very justly remarks that another strong reason of the difficulty of diagnosis is found in the fact that no two epidemic or atmospheric diseases can possibly prevail together without becoming blended. Such, as we have seen, is the case with respect to febrile complaints, though particularly to common autumnal periodic fevers. When these amalgamations or complications occur, we can readily conceive that the physician, unless well skilled in the matter, and careful in his mode of observation, is liable to commit great errors of diagnosis, mistaking one disease for another, or concluding—as every one has known to occur—that he has before him a disease very different from any he has heretofore seen or heard of. When, however, yellow fever assumes a decidedly epidemic form, or there does not exist at the same time a strong antagonistic diseased force to mar the purity of its features, it exercises full sway, and takes entire possession of the field. As Dr. Nott remarks, the peculiar characteristics of the disease stand out in bold relief, and, with few exceptions, all difficulty of diagnosis vanishes. Patients

¹ New Orleans Journal, iv. 584.

are stricken down in greater number with attacks varying from the mildest to the most malignant, and yet all wholly unlike other febrile diseases, and all presenting groups of phenomena which plainly indicate the identity of their nature.

The physician who would be astonished at the appearance of black vomit, in a case of what he fancies to be ordinary fever, and who would always wait till the occurrence of that formidable symptom before establishing his diagnosis and pronouncing the disease to be yellow fever—who could not know the latter to be such till the patient was *in articulo mortis*—should be advised (supposing the case to have been really of the kind mentioned—for every one knows that black vomit alone is not sufficient to characterize yellow fever) to go back to school, or to keep his eyes wider open the next time. If the appearance of black vomit were indispensable to enable the physician to establish his diagnosis, it would follow that, in a large proportion of cases of what there can be no doubt is yellow fever, the true nature of the disease could not be positively ascertained. As a general rule, it may be stated that, in cases that recover, black vomit does not make its appearance. In this city, the mortality among the reported cases has averaged one in 2.12, the proportion varying from one in 1.2 to one in 3.86. In other places, the loss has occasionally been less. The number of patients who escape that symptom must hence be everywhere larger than that of those who suffer from it. The amount of the former will be found to be even greater than here stated; for those who die do not all eject the fluid, and it then requires an autopsy to ascertain that it has been effused. And yet no difficulty is experienced, in individuals who recover, or die without throwing up the black matter, in distinguishing the disease from other forms of febrile complaints. Such errors are generally the result of want of skill or want of attention on the part of the observer; for the yellow fever, and also the remittent, present, together with phenomena approximating them to each other and to different complaints, characteristic features of their own, which, when duly and carefully examined and analyzed, prevent the one from being mistaken for the other, except in cases of an anomalous or complicated nature, or at the very outset of the attack—of mild cases especially—or which, at any rate, enable the physician to establish his diagnosis, in the ordinary forms of the disease, long before the accession of that formidable symptom. The blunders of the unskilful or careless, or the fancies of the unitarian, must not be urged in denial of the possibility of tracing a line of distinction between the different forms of autumnal fevers. Let them, by way of encouragement, peruse the following statement, made by a clever physician of Mobile: “Two clergymen of this city, Mr. Balzan and Mr. Dorman, whose active benevolence has won for them much correct information in relation to fevers, have frequently, in my private and hospital practice, designated each variety and grade of fever with the greatest ease and correctness, separating the grave from the ephemeral cases of yellow fever, and distinguishing these again from periodic fever. In their diagnosis they were governed by the character of fever, pain,

restlessness, colour of skin, physiognomy, paroxysms, and nature of the secretions."¹

Easy, however, as it is, usually to discriminate between yellow fever after it has fairly set in and other forms of febrile complaints—a few obscure and rapid cases, perhaps, excepted—it approximates so closely, in a greater or less number of points, to several diseases, as to render the diagnosis sometimes embarrassing to the less expert, and even to lend support to writers who regard it as identical with, or as a modification of other fevers usually held as distinct, while, on the other hand, some forms of fever which are viewed as simple varieties of the yellow fever, have been held up as separate and distinct complaints, arising from different causes, located in different climates, characterized by different phenomena, and governed by different laws. Such being the case, so respectable is the professional character of many of those who entertain the opinions mentioned, and so convincing have the reasons assigned in their support been considered, that it becomes proper to examine into their merits, and, by indicating the points of resemblance or dissimilarity presented by those several diseases, to endeavour to arrive at a definite conclusion on the subject of their diagnosis, and of the relationship existing between them.

By some writers in this country and elsewhere, attention has been especially called to the great analogy supposed to exist between the yellow fever and the oriental plague; and while some have discovered in these symptoms only a proof of affiliation, indicating their belonging to the same family of diseases,² others maintain that those diseases approximate so closely to each other, in regard to their causes, to the phenomena they exhibit, and the treatment they call for, as to render the conclusion inevitable that they are, to all intents and purposes, the same complaint, though modified, more or less, by diversity of climate and of the temperament, constitution, and mode of living of the individuals attacked.

The opinion was entertained, more than a century ago, by Dr. H. Warren,³ who, as already stated, fancied that the yellow fever of Barbadoes had reached there in some roundabout way from the Levant. In this country, the identity in question was originally suggested by the late Dr. E. H. Smith, of New York, in his *Essay on the Plague of Athens*,⁴ and subsequently maintained, more or less elaborately, by the late Dr. Caldwell and others, in Philadelphia and elsewhere.⁵

If, as is argued by the advocates of this opinion, with a knowledge of

¹ Lewis, Fever of Mobile in 1847, N. O. J., v. 40.

² Monges, ii. 62; Robert, p. 360, &c.; Dalmas (1st ed. p. 60); see Med. Repository, iv. 400.

³ Warren, A Treatise concerning the Malignant Fever, &c., pp. 3, 5.

⁴ Med. Repos., i. 28.

⁵ Caldwell, An Address to Philad. Med. Society on the Analogy between Yellow Fever and true Plague (1801) pp. 7-30; Jenks, an Essay on the Analogy of the Asiatic and African Plague and the American Yellow Fever, Philad., 1804, p. 13; Boisseau, *Pyréto-logie*, pp. 445-7; Audouard, *Fev. Interm. Pernicieuses*, p. 57; Kelly, xiv. 387.

what has been said of the yellow fever, we consult the many works written on the plague in olden and modern times, the analogy, on all the important points, will appear manifest.

1. Both diseases are located in cities, seldom in villages, and not in country situations.
2. They appear at the close of spring, or in the course of the summer, reach their height in autumn, and decline and disappear on the accession of cold weather.
3. They occur epidemically at certain irregular periods, or after indefinite intervals of time.
4. When they appear epidemically, they reign supreme, take the place of other diseases, or impress their livery upon them.
5. They are preceded, followed, or accompanied, in adjacent places, either by new diseases or by an increase in the frequency, but more particularly in the malignancy, of common diseases.
6. They are of a more fatal tendency at the onset of an epidemic.
7. They always appear in places calculated to give rise to an abundance of putrid exhalation.
8. Their progress is too rapid to depend on a cause of such limited power as contagion.
9. Neither are communicated away from the limits of a malignant atmosphere.
10. They attack the same class of individuals.
11. They attack the same species of inferior animals.
12. Their appearance is marked, in the place they visit, or in adjacent localities, with similar peculiarities in the surrounding and concomitant phenomena of nature.
13. They are both ushered in by febrile phenomena, and in both, cases occur in which there is an absence of febrile excitement—the pulse and heat of skin being natural.
14. They both direct their principal force against the head, stomach, and abdominal viscera.
15. While the black vomit, the principal characteristic of the yellow fever, sometimes fails in that disease, it sometimes occurs in the oriental plague.
16. Buboes and carbuncles, the main characteristics of the plague, occasionally show themselves in the yellow fever.
17. In some cases of plague, a yellow suffusion, similar to that of yellow fever, occurs.
18. In plague, the muscular strength is sometimes retained; the tongue is sometimes natural, at others white, with rosy edges and tip; the patient complains of a burning heat at the epigastrium, and pain in the loins and forehead; the eye is brilliant and phosphorescent, and the patient complains of a feeling of anxiety.
19. Both have morning remissions and evening exacerbations, though more frequently in the plague than in the yellow fever; and the metaptoxis, so generally encountered in the latter, is sometimes observed in the former.
20. Both have the same critical movements.
21. Both prove destructive, sometimes, in a few hours.
22. In general, neither is so rapid—generally reaching their crisis on the third, fifth, or seventh day, sometimes on the ninth or eleventh.
23. They resemble each other in their decline and termination.
24. The reasons assigned against or in favour of the contagious character of the plague are the same as those adduced against or in favour of the contagiousness of the yellow fever.
25. In both, the anatomical characters are often much the same.¹

¹ See on the subject of the plague, Hodges, *Loiomologia*, pp. 49, 85, 98; Pugnet, *Obs. sur les Fièvres Pestil. et Insidieuses du Levant*, p. 152, &c.; Faulkner, *On the Plague of*

Close, however, as the resemblance undoubtedly is in some cases, we may well doubt whether it is sufficiently so to justify our regarding the yellow fever and the plague as mere modifications or degrees of one and the same disease. It is to be borne in mind that some of the symptoms mentioned as common to both are encountered just as often in other febrile complaints, between which and the yellow fever scarcely any analogy exists, as in the plague; and that, hence, their occurrence in the latter cannot be adduced in proof of its identity with, or very close analogy to, the former. These symptoms belong to all febrile complaints, and are not typical of either in particular. Nor is this all. Some of the phenomena of plague, which, while not observed in ordinary fevers, are noticed in the yellow fever, and some of those of the latter, that are seen in the plague, will, on examination, be found to occur so seldom in both instances, as scarcely to deserve being considered as the result of anything more than accidental complications, similar to those observed in the most dissimilar complaints. Buboes, for example, are undoubtedly met with in the yellow fever; but they are so only in a very limited number of cases—sometimes once or twice during the course of a whole season, sometimes not even in a single instance. In the plague, on the other hand, though, according to Russell (pp. 96, 112; *Chit.*, pp. 30, 31) and others, they sometimes fail to appear, this failure is of most rare occurrence, and is usually noticed in cases ending rapidly in death. In the vast majority of instances, they show themselves from the second to the fourth day, in the axillæ, groin, or neck, and sometimes constitute, even in mild attacks, one of the earliest symptoms of the disease. In not a few it is the only one noticed. Buboes, in fact, are characteristic of the plague, occurring in those that recover and those that die. The same remarks are applicable to carbuncles, which very seldom occur in the yellow fever, and to petechiæ, which are very often absent; but are, on

Malta, Edinb. Journ., x. 143; *Ib.*, Treatise on the Plague, p. 221; Sydenham, i. 97 (Syd. Soc. edit.); Diemerbroeck, Tractatus de Peste (1665), iv. 97, &c.; Deidier, Traité de la Peste (1744), p. 4; Semoilowitz, Mém. de la Peste qui en 1771, ravagée l'Empire de Russie (1783), p. 132, &c.; Russell, A Treatise on the Plague, p. 80, &c.; Aubert Roche, De la Peste ou Typhus d'Orient (1843), p. 250, &c.; Chicoyneau, Traité des Causes, des Accidens, et des Cures de la Peste, p. 41; *Ib.*, Lettre pour prouver, &c. (1721), p. 3; Mead, A short Disc. concerning Pestilential Contagion, &c. (1720), p. 2, &c.; Larrey, Campagnes d'Egypte, p. 317; Brayer, Neuf Années de Séjour à Constantinople, ii. 249; Cholet, Mém. sur la Peste, p. 92; Boyer, Diss. Abrégée sur la Maladie de Marseilles (1721); *Ib.*, Réfutation des Anc. Opinions touchant la Peste (Paris, 1721); Wilson, A Treatise on the Plague, &c., p. 71, &c.; Hecquet, Traité de la Peste (1722), p. 118; Agnes, Diss. Abrégée sur la Peste de Provence Vienne (1721); Pestalozzi, Opusculs sur la Maladie Contagieuse de Marseilles (Lion, 1723); Chicoyneau, Verney, and Soullier, A Succinct Account of the Plague of Marseilles, from the French (London, 1721); Bradley, The Plague at Marseilles considered (London, 1721); Desgenettes, Hist. Méd. de l'Armée d'Orient (Paris, 1802); Bertrand, A Historical Relation of the Plague at Marseilles in 1720, from the French (London, 1805), p. 29, &c.; Morio, Storia della Peste di Noja, Napoli (1817); Clot-Bey, De la Peste Observée en Egypte, pp. 29, 36, &c.; Rapport à l'Académie de Méd. sur la Peste (Paris, 1846).

the contrary, almost constant attendants on the plague. On the other hand, the black vomit, so frequently encountered in the yellow fever, is but very rarely, though sometimes, met with in the plague. It may be doubted, from the imperfect accounts that have as yet reached us on the subject, whether, in all the cases in which it is said to have occurred, the matter ejected was of the same nature as that thrown up in the other disease. Jaundice, also, is represented as a symptom of rare occurrence in the plague, in the last and malignant form of which, the skin assumes not a mahogany, but a *cyanose* hue. Epistaxis, hæmatemesis, and hæmaturia are also represented as occasional symptoms. Pain in the limbs is a rare attendant on the plague; nausea, in the same disease, has been found a more common symptom than vomiting, which is absent in a large proportion of cases. The metaptoxis so characteristic of yellow fever, is not reported as common in the plague; and, when occurring, is followed, unlike what takes place in the other, by exacerbations of fever. The eye, in plague, though occasionally brilliant and phosphorescent, is more frequently muddy and dull, and red only during the exacerbation. The bowels are less generally costive in the plague, and the costiveness, when it occurs, is not detrimental, however long continued; while looseness is always dangerous, unless all other signs are favourable. In a word, while jaundice, the black vomit, the red, fiery, and humid eye, and the single paroxysm are seldom seen in plague, they are characteristic of the yellow fever; while buboes, carbuncles, and petechiæ, which are not generally seen in yellow fever, are characteristic of plague. To these points of difference, which establish a broad line of demarcation between the two diseases, may be added the following:—

1. In countries subject to the plague, there is no acclimatization against its attacks, constant residence or nativity affording no protection, as is the case, in hot climates, relative to the yellow fever. Indeed, the disease bears more heavily on natives than on Europeans, however recently these may have arrived in the country. Pugnet, it is true, remarks, in relation to the epidemic of Damietta, that not only had the disease become concentrated within the walls of the city, but prevailed only among the Franks and Greeks (p. 176). In general, however, the reverse is the case. Sir Robert Wilson says that in Egypt, while the natives were severely affected, British soldiers might hold free intercourse with the infected.¹ Bancroft says, also, that some of the native Indians were affected, but the British soldiers were not (p. 581).

2. The plague, unlike the yellow fever, is very prevalent among negroes, and gives rise among them to a larger amount of mortality than among whites. The same result obtains among East Indians. This was found to be the case in the British East Indian army in Egypt (*Bancroft*, p. 579). Bancroft also mentions the greater prevalence of the disease among negroes (p. 579). So do Desgenettes, Pugnet, Satira, and Clot Bey. The latter states that at the outbreak of the epidemic of 1835, out of more than five

¹ Hancock, *Researches into the Laws and Phenomena of Contagion*, p. 190.

hundred negroes that were at Cairo, all but eighteen died; of sixty in one family, fifty-four perished.¹

Plague attacks females more than males, and, in general, children also;² feeble and nervous constitutions are more prone to it than robust and sanguine—but the disease in these is more severe and fatal. From this it would appear that the Eastern disease attacks classes of people different from those which suffer from the yellow fever. The difference in that respect does not depend upon a difference of the nationality of the individuals affected by the two, for the same results are obtained in Egypt and elsewhere when the plague attacks Europeans, and in Europe the disease manifested the same predilection as it does now in the East.

3. The plague, though generally occurring principally in cities, is not exclusively limited to them; for in Egypt and elsewhere it often extends to villages, hamlets, or even isolated houses at some distance from these. With few exceptions, the contrary has been found to hold in regard to yellow fever. Though evidently originating—at least commonly—and prevailing amid sources of infection, it has shown itself amid the high, arid, and barren of Syria, as well as along the canals, &c., of lower Egypt.³

4. It has been stated that the plague, like the yellow fever, prevails usually in summer, and is put a stop to by cold weather. Such has been the case in Russia, Poland, Great Britain, France, and some parts of the East.

At Marseilles, the disease broke out on the 20th of June, ran till the 1st of July, raged through August and September, declined in October and November, and ceased in winter. In London, after some cases in November, December, and February, it broke out in May, continued violently till the 10th of September, then declined till December, when it disappeared. “Like everything else, too, in nature,” says Sydenham, in speaking of the plague, “it has its proper periods of increase and decline; it takes birth at the

¹ De la Peste Obs. en Egypte, p. 112.

RACES IN ALEXANDRIA, 1834.

Europeans—English, French, Russians, and Ger-

mans	52 in	1,000,	5.2 per ct.
Italians	118 in	1,600,	7.3 “
Turks	678 in	6,000,	11.3 “
Copts, Jews, and Armenians	482 in	4,000,	12 “
Greeks	257 in	1,800,	14.2 “
Arabs (soldiers)	470 in	3,000,	15.6 “
Arabs (civilians)	10,936 in	20,000,	54.6 “
Maltese	367 in	6,000,	61 “
Negroes and Barbarians	1,528 in	1,800,	84.9 “

[Aubert Roche, p. 25.]

SMYRNA, 1837.

Turks and Jews	17	per cent. of population.
Catholics (Greek) and Europeans	0.8	“ “ “
Armenians	6.3	“ “ “
Greeks	1.9	“ “ “

² Clot Bey, op. cit., p. 7.

³ Bancroft, p. 519.

period given above (when spring passes into summer), and it rises towards maturity as the year advances; with the decline of the year it declines also. Finally, the frosts of winter transform the atmosphere into a state unpropitious to its existence."¹ At Erzeroum, the capital of Armenia, the disease, in 1840, broke out in June, and in 1841, in August. In both instances, it was put a stop to by the severe cold of winter.² The plague epidemics of Constantinople invariably commence in the summer season, and are effectually cut short by frost.³ Volney, who, among others, has noted the fact of the cessation of the disease on the accession of winter, says, properly: "The winter destroys the plague at Constantinople, because the cold is there very severe. The summer lights it up, because the heat is damp." In Russia and Poland, the same result has been noticed. If in Egypt, and other hot latitudes, the disease usually prevails in winter—commencing in November and ceasing in June—it is because that season is warm and damp. But even there, as we gather from no less an authority than Desgenettes,⁴ while the south winds, as well as hot and damp air favour, if they do not occasion its development, the north winds, and the extremes of cold or heat, put an almost complete stop to it. Much the same statements are made by Larrey,⁵ Clot Bey,⁶ Assalini,⁷ and Pugnet.⁸

Mr. E. Robertson, who, during a long stay in Syria, paid much attention to the subject of the etiology of the plague, says that the disease in Turkey and Lower Egypt can only exist in a temperature between sixty and eighty degrees (Fahr.), "a lower or higher either modifying or utterly destroying that atmospheric constitution, or those other occult causes, giving rise to its origin and propagation."⁹ Nor can the occasional continuance of the plague at Aleppo, as we learn from the admirable accounts of the disease handed down to us by Drs. Alexander and Patrick Russell,¹⁰ through the winter, after the accession of frost, and even a fall of snow, be cited as militating against the view here maintained. Such instances of continuance were isolated and exceptional. In general, the disease has there ceased before the accession of winter—often during the months of August, September, and October—having begun to decline in July; and frequently it has ceased later on the appearance of cold. In those instances, as in 1762,¹¹ when different results were obtained, the cases were few in number, and it is not unreasonable to attribute them partly to the latency of the poison in the sys-

¹ Vol. i. p. 100, ed. of Syd. Soc.

² Report of Academy of Medicine, p. 31.

³ Brayer, *Neuf ans à Constantinople*, ii. 77; Clot Bey, *Traité de la Peste*, p. 225; *Rapport fait à l'Acad. Roy de Méd.*, p. 330.

⁴ *Hist. Méd. de l'Armée d'Orient*, p. 248.

⁵ *Campagnes d'Egypte*, p. 330.

⁶ *De la Peste Observée en Orient*, p. 266.

⁷ *Observations on the Disease called the Plague, &c.*, p. 42.

⁸ *Mém. sur les Fièvres de Mauvais Caractère, &c.*, p. 204.

⁹ *Medical Notes on Syria*, *Edinburgh Journal*, lxii. 331.

¹⁰ *The Natural History of Aleppo*, 2d ed. iv. London, 1794; *A Treatise on the Plague of Aleppo in 1760, 1761, and 1762*. London, 1791.

¹¹ *A Treatise, &c.*, p. 44.

tem of persons exposed to its influence previous to the change of weather, and partly to the short continuance of a temperature sufficiently low to destroy completely the poison, which broke out anew on the accession of heat.¹ It is true that, in the neighbouring villages, caves, and grottos, the disease continued some time in winter, after having ceased in the city; but in these places it showed itself exclusively among the poorer classes, whose habitations, from the mode of their construction, their want of proper ventilation, the filthy condition in which they were kept, and the great heat in which their inmates indulged, may well be supposed to have retained the poison in sufficient force, if not to have been hotbeds of the pestilence, during the comparatively moderate and short winter of that country, where, according to Dr. A. Russell, the thermometer, in a series of nine years, varied in October, from 51 to 84; in November, 44 to 65; in December, 40 to 55; in January, from 34 to 57; in February, from 48 to 55; in March, from 44 to 67; in April, from 56 to 82; while in May it mounted up from 67 to 92. To this it may be added that, in most places where the plague has prevailed during the winter months, as in Egypt, at Malta, Toulon, Aix, Venice, Messina, &c., that season is not characterized by severe or long continued cold weather, and a hard bleak frost is seldom if ever experienced.

But, on the other hand, the plague has never prevailed between the tropics,² where heat and various sources of infection abound; nor does it do so in our climate. As is known since the days of Prosper Alpinus, it never begins in Egypt before the month of December, nor later than March, and ceases in June on the accession of the hottest weather.

The epidemics of Stockholm, Dantzic, and Hamburgh appeared at the close of the year prior to their assuming the epidemic form. In London, in 1625, one parish was infected as early as January. The same may be said of the plague of Nimeguen, Cronstadt (Transylvania), in 1736, and Vienna in 1713. In each of these, a short interval of suspense occurred during the heart of the winter; but the disease prevailed in cold weather. Chenot says: "Observation has taught us in Europe that if a plague begins in Spring and summer, it ceases the following winter—but if it first appear in autumn, it is protracted to a much longer period" (p. 32). Diemerbroeck says: "A winter plague is more violent and of longer duration than one which begins at any other time" (cap. 3).

At Toulon, in 1720, the disease broke out in the beginning of October, continued through the winter, and ceased on the 18th of August, 1721. At Aix, it began in August, 1720, and ceased on the 12th of July, 1721. At Montpellier, in 1629, it commenced in July, was most violent in October, November, and December, gradually diminished till April, when it disappeared. In Milan, it prevailed from the end of October to the close of December, 1630. In Lyons, 1628, '29, it began at the end of September, abated in March, 1629, and nearly ceased in July; but it recommenced in August, and disappeared in September of the latter year. In Smyrna, the

¹ A Treatise, &c., p. 45.

² Bancroft, p. 579.

plague commences in October or November. If the epidemic is strong, it continues all winter, goes on increasing till May, then diminishes and ends in August, on the 15th of which the quarantines are removed.¹ It has been adduced, in explanation of the difference on that score between plague and the yellow fever—which has never been known to occur in winter and stop in summer—that the heat of an Egyptian winter is equal to that of the summer season here, and other yellow fever countries; that in other places where the plague has prevailed during the winter, as Athens, Malta, Toulon, Aix, Venice, Messina, &c., that season is mild, and not characterized by severe or long-continued cold weather, and that there a hard black frost is seldom if ever experienced; while the cessation of the disease in some of them in June is due to the destruction of the exhalations producing the disease, consequent on the excessive dryness which ensues. But, in answer, it is remarked that, at Aleppo and other places, the plague spread while the earth was covered with snow—that in many cities if snow did not exist, the disease certainly prevailed during cold, and even *very* cold weather—therefore, during a state of temperature very different from that required for the outbreak and spread of the yellow fever. In view of what precedes, we may say that everything justifies the conclusion, that so far, at any rate, as relates to such European countries where epidemic plague has shown itself, it has been found to commence, spread, and cease at all seasons of the year,² and under very dissimilar conditions of temperature—a circumstance not observed to hold in relation to the other disease.

Cholera.—Dr. Robert³ regards the cholera asphyxia of India as a modification of the yellow fever of tropical regions produced originally by the same morbid cause; giving rise to analogous symptoms, occurring and disappearing at the same seasons of the year, and presenting kindred analogous characters. Other physicians, in Europe and this country, regard the cholera as a form of ordinary autumnal fevers; among which some of them include the yellow fever.⁴ The idea is not new; for Dr. Pariset at one time thought that they were convertible diseases, suggesting as very possible that the yellow fever of Cadiz, in 1819, was the effect of the cholera, imported from Calcutta, and which, during the passage from India, had lost its pristine characters and assumed those of the other disease.⁵

But whether new or old, the opinion will not stand the test of examination; for, on examination, we cannot fail to discover that the two diseases no more resemble each other in respect to phenomenal and anatomical characters, mode of progression, and laws by which they are governed, than do small-pox and measles. If they approximate on any point it is in appearing with greater force in localities noted for certain sources of malarial infection. But from this circumstance, no argument in favour of identity, pathological

¹ Aubert Roche, p. 11.

² Report of Acad. of Medicine, p. 59.

³ Guide Sanit. des Gouv. Europeens, &c., pp. 48, 365, 367, 368, 373.

⁴ Boudin, Bertullus, Merrill, Searl, Maréchal, Mouchet, &c.

⁵ Observations, &c., pp. 56, 59.

and etiological, can be derived, for the attentive inquirer will not be slow in perceiving that in the case of cholera, as in that of some other zymotic diseases which have nothing in common with the yellow fever except their toxic origin, the effluvia arising from these foul localities, as well as putrescent food, impure water, &c., produce their deleterious effects, not by furnishing the efficient cause of the disease, but by predisposing the system of those exposed to their influence to receive the morbid impress of that cause, or excite it into action.

The cholera is not, like the yellow fever, a disease limited to a certain zone, and attacking chiefly those who, from northern climates, have recently arrived within that zone, or near its limits. So far from this, it extends sometimes over a large surface of country—spreading from one continent to another, and ravaging the natives of all climes. It is not confined to tropical and equatorial regions, characterized by high thermometrical heat; but spreads not only to these but to others of a diametrically opposite character. It acknowledges no acclimatization, attacking everywhere, with equal force, natives and new comers; and showing no preference for the inhabitants of northern climates. Though it prevails generally and more violently in localities similar to those in which the other originates and spreads, it affects such localities not in cities or small portions of these only, but penetrates to remote and inland situations, where the fever has never, and probably never will be known; and even in detached residences remote from accumulations of population. Its mode of progression is very different; flying from one part to another in a most rapid, capricious, and inexplicable manner—generally in a westerly direction. Though generally appearing in summer, and ceasing on the accession of cold, it disappears often without the appearance of the latter, and during the continuance of atmospheric conditions similar to those under the influence of which it appeared; while, in some instances, it has broken out and prevailed violently during the cold weather of a Siberian winter.

If, in respect to some of its symptoms—the colour and consistence of the blood, and the usual condition of the intellectual faculties, &c.—cholera approximates to the yellow fever, every one knows that in relation to the greater number—and those the most important—it differs from it very materially.

The vomiting and purging of rice-water fluid, the excruciating muscular spasms of the extremities and trunk, the corrugated appearance of the fingers, the cold tongue, the cyanose hue of the skin, so characteristic of cholera, and so constantly absent in the yellow fever; and, on the other hand, the jaundice, black vomit, and other symptoms peculiar to the yellow fever, and so rarely, if ever seen in cholera; all these circumstances, together with the total absence of fever in the latter, until, sometimes, at an advanced period, when reaction has taken place, point out the existence of a broad line of demarcation between the two diseases, and lead to the belief of their distinct and specific nature.

Causus, or Inflammatory Endemic.—By many writers, Towne, Moseley, Desperrières, Miller, Gillespie, Dickinson, Veitch, Copland, Thomas, and

Valentin, the yellow fever has been assimilated to the inflammatory form of fever—the *causus* of the older writers, and *synocha* of Cullen. By some of these, the latter, as it appears in tropical climates, has been described under the name of the former; while, by others, the name of *causus* has been assigned to some forms of true yellow fever. There can be no doubt, that, in some of its varieties—the inflammatory—the yellow fever bears a marked resemblance to the *causus*. But that resemblance exists principally during the first stage of the attack, and ceases, or lessens greatly as the disease advances, the true *causus* being but seldom attended, except under circumstances of complication, with the symptoms characteristic of the last stage of yellow fever. Unlike the yellow fever, which is the offspring of local and terrestrial, and not general influences, the *causus* is the product of the direct action of heat, particularly of insolation—or of any cause exciting vascular irritation in a system predisposed to morbid impressions. It prevails in all localities where the temperature is high, and the atmosphere dry, and is very frequently encountered in places where pure yellow fever has never appeared, and which are perfectly unsuited to its production. It attacks less suddenly and unexpectedly. The pain of the head, by which it is attended, is much more diffused, and not restricted to the frontal or temporal regions—extending from the occiput to the neck, and even lower. It is characterized throughout its course by distinct evening exacerbations and morning remissions, especially on odd days—a circumstance seldom observed in the pure yellow fever. It never presents the metaptosis characteristic of the latter, and during which there is a lull of all the symptoms; but passes, when not arrested by proper treatment, from a stage of high excitement to one of prostration, attended with a continuance or aggravation of dangerous symptoms; while in mild or curable cases the fever subsides gradually. The *causus* is attended very generally, if not always, with evident affection of cerebral organs—contraction of the pupils, subsultus tendinum, carpalgia, delirium, and convulsions—followed by somnolency and intense coma. In the yellow fever, these symptoms form no essential part of the disease, and when they do occur, are generally the result of complications, such as may occur in all inflammatory or other complaints. The patient, when delirious in the *causus*, remains so, and does not regain possession of his intellectual functions and retain them to near the closing scene. Soon after the close of the state of calm in yellow fever, the patient is affected with continued nausea and vomiting, hiccup, hemorrhages from different outlets, excessive pain and sense of ardor at the epigastrium and umbilicus, painful respiration, sense of anxiety, and deep sighs. His physiognomy generally expresses pain and despair. His skin is generally jaundiced, and he as generally throws up from the stomach, or passes off by stool, large quantities of black matter. All these phenomena are foreign to the pure *causus*; and if, in some cases, they are observed, the event may be traced to accidental complications—not unusual—especially the jaundice—in diseases of hot climates. After death, the brain and membrane are found principally implicated, and the digestive organs secondarily so in *causus*; the reverse takes place in yellow fever.

Scurvy.—By some writers, Dalmas, Foureau de Beauregard, Bertulus, Vatable, and Hombron, in Europe, and Prof. Senac, of New Orleans, in this country, great analogy is thought to exist between the yellow fever and scurvy. This opinion is principally founded on the hemorrhagic tendency manifested in both, the existence of ecchymosis, the adynamic symptoms presented in the latter stage of the yellow fever, and approximating to those of the scurvy; and on the supposed beneficial effects in the former of remedies endowed with antiscorbutic effects.

To these points of approximation, Mr. Bertulus adds the deteriorating nature of the causes, giving rise to the two diseases. "It is thus," he remarks, "that scurvy, which we regard as principally due to the use of insalubrious aliments producing materials of an unhealthy character, and incapable of repairing the losses of the system, is strikingly analogous to certain miasmatic affections. Here the morbid agents are evidently of a different nature; but they have for their common effect the general intoxication of the economy. In the yellow fever, plague, &c., this intoxication takes place in a few days, and even hours. In scurvy, it is produced gradually and very slowly—insomuch, indeed, that we may, to a certain extent, regard the disease as the chronic state of intoxication of the economy by morbid principles" (p. 53).

But, even admitting that the analogy in these respects were even closer than it will be found to be on inquiry, few physicians, conversant with the two diseases, will feel prepared to admit, on that account alone, their identity, nor will they experience much difficulty in establishing their diagnosis. Unlike the yellow fever, which attacks suddenly, affects principally individuals in the vigour of life and of sanguine temperament, runs its course in a few days, and often sets in with symptoms of inflammatory reaction, both general and local—scurvy is never, unless under circumstances of complication, attended with febrile reaction, or inflammation of internal organs; it makes its approach in a gradual manner; may last weeks or months, and even leave a taint behind difficult to eradicate; usually appears in climates or seasons, and under the influence of meteorological agencies different from those that have been pointed out in relation to the yellow fever. It principally affects individuals already enfeebled by privations, improper and innutritious food, and calls for a plan of treatment totally inappropriate to the case of the other.

Typhoid Fever.—From this form of febrile disease, between which and the yellow fever some medical writers have fancied they discovered points of analogy sufficiently strong to justify a belief in their identity, the latter may, without difficulty, be distinguished. The long duration of the former—extending, sometimes, over a period of six weeks, or even sixty days—its occurrence, often, in country and other situations where, and in seasons of the year when, the other never has shown and never can show itself, must alone serve to point out a difference between them. Typhoid fever is a disease of temperate climates, prevails in all seasons, is not arrested in its course by meteoric agencies that affect the yellow fever, and, unlike the latter, selects its victims among individuals of weakly constitutions, and who have suffered

from insufficiency of food. The purple flush, and, in its absence, the dusky hue of the face; the obtuseness, heaviness, and dulness of the countenance; the tendency to remission once or twice a day; the frequent occurrence of diarrhœa, even from the commencement of the attack; the equal frequency of a greater or less degree of tympanitis; the rose-coloured eruption, which is an attendant on almost every case, and which appears during the first week of the attack; the sudamina, which appear somewhat later; the hebetude of mind, followed by a mild delirium; the hardness of hearing, amounting, sometimes, to complete deafness; the cough and bronchial râle; the frequent occurrence of nervous symptoms, subsultus tendinum, twitching of the face, and epileptic spasms; the retention of urine; the discovery, on dissection, besides inflammatory congestions in various organs, of morbid changes in the Peyerian glands; all these phenomena, which are peculiar to the typhoid, and seldom, if ever, noticed in the yellow fever—together with the absence of the metaptoxis, and of the subsequent collapse or freedom from febrile excitement; the absence, too, of jaundice, and of the ejection from the stomach of black matter, of yellow discoloration and fatty degenerescence of the liver, enable us to draw a line of demarcation between the two diseases.

Sweating Miliary Fever.—A parallel has been drawn between the yellow and the sweating miliary fever of France and other countries; and not a few professional writers have strangely enough concluded that the two diseases are the *analogues* of each other—in other words, analogous in respect to nature and causation, but differing only as regards peculiar phenomena resulting from the diversity of climates in which they occur; the one disease belonging to southern, and the other to northern latitudes. M. Rayer, in a work of great learning and ability on the sweating fever which prevailed in the Departments of Oise, and Seine and Oise (France) in 1821,¹ has drawn the following parallel:—

<i>Yellow Fever.</i>	<i>Sweating Disease.</i>
The disease appears in humid localities.	The disease appears in humid localities.
<i>Symptoms.</i> —Pain, and uneasiness at the epigastrium, eructations, nausea, and vomiting.	<i>Symptoms.</i> —Pain, uneasiness at the epigastrium, sometimes eructations, nausea, and vomiting.
Difficulty of breathing, sighing, oppression, epigastric palpitations.	Difficulty of breathing, sighing, oppression, epigastric palpitations.
Uneasiness, anxiety, agitation, wakefulness, or coma.	Uneasiness, anxiety, agitations—sometimes coma.
Determination to the skin, with subcutaneous ecchymosis and yellowness.	Determination to the skin, with continued sweat, and a miliary eruption.
Delirium.	Sometimes delirium.
Dysuria, and suppression of urine.	Sometimes dysuria.
<i>Anatomical Characters.</i>	<i>Anatomical Characters.</i>
Inflammation of the stomach, of the gall-bladder, and sometimes of the brain.	Inflammation of the stomach, and sometimes of the brain.

¹ Hist. de l'Epid. de Suetie Miliare qui a Régné en 1821, &c., p. 462, &c.

When we bear in mind that many of the symptoms which, in the foregoing parallel, are represented as common to both diseases, are not less so to other complaints of dissimilar character, and cannot therefore be regarded as establishing an important analogy between the two former; when we remember that while some are stated to be constantly seen in the one, they are admitted to occur only sometimes in the other; and that nothing can be more dissimilar than the subcutaneous ecchymosis and jaundice of the one, and the profuse and continued sweat, and the miliary eruption of the other; when, with this, we observe that no mention is made of the nature of the matters ejected from the stomach and discharged from the bowels—which every one who has investigated the subject must know to differ materially in the two diseases—or of the mode of succession of the symptoms in, and of the type of, both fevers, and of the hemorrhagic tendency in the one, and its absence in the other, we can have no difficulty in perceiving that the analogy thus proclaimed to exist between the yellow and sweating fevers, is not sufficiently striking to incline us to the belief that they are only different forms of one and the same disease—identical as regards nature and cause, but modified by differences of climate.

If we examine the various descriptions we possess of the sweating disease, from the day the disease first broke out among the troops of Henry VII.—those of Caius,¹ of Tessier,² Saint André,³ Boyer,⁴ Rayer,⁵ Dubun de Peyrelongue,⁶ Parrot,⁷ Gaillard,⁸ Andry, Jeanroy, and Poissonnier,⁹ Pujol,¹⁰ Colson,¹¹ Foucart,¹² Rayer,¹³ and Bricheteau¹⁴—we find nothing which can, without the aid of imaginary powers of great amplitude, be construed into a resemblance to the yellow fever. In the disease described by Caius, and which, from its most prominent symptom, and its being supposed to be peculiar to England, has received the denomination of *Sudor Anglicus*, the patient, after a few premonitory symptoms of no great importance—generally without any warning—was seized with a profuse perspiration, internal heat, intense thirst, agitation, delirium, sleepiness, sometimes permanent loquacity, languor and inertia of stomach, vomiting, præcordial palpitations and anxiety, headache, strong, quick pulse, quick, short, and laborious respiration, red and purplish spots over the body, transparent water-blisters over the neck, in the axillæ, on the chest, and abdomen. At other times, a

¹ De Ephemera Britannica (Londini, 1721), p. 23.

² Mém. sur la Suette qui a régné à Hardivilliers en Picardie, Mém. de la Soc. Roy. de Méd., ii. 46.

³ Topogr. Méd. du Dep. de la Haute Garonne, p. 8.

⁴ Méthode à suivre dans le Traitement des Mal. Epid. Paris, 1762.

⁵ Op. cit., p. 48.

⁶ De l'Epidémie qui à régné, &c., en 1821, dans la Dep. de l'Oise, &c. Paris, 1824.

⁷ Mém. de l'Acad. de Méd., x. 394.

⁸ Considération sur l'Epidémie de Suette Miliare qui à régné à Poitiers (Paris, 1845), p. 8.

⁹ Méd. Eclairée par les Sci. Phys., ii. 47.

¹⁰ Œuvres Complètes, iii. 294, &c.

¹¹ Bulletin, xiv. 679.

¹² Ibid., p. 925.

¹³ Ib., vii. 57, 188–9.

¹⁴ Mém. de l'Acad., p. 9.

miliary eruption spread over the body. These symptoms ran their course in a few hours. In the disease, as it has frequently appeared in France during the last hundred years, the symptoms are much like those just described, but of slower progress. The sweat is most profuse, and of an insipid and nauseous odour. The patient complains of pain in the lower extremities; the conjunctiva is humid, seldom injected, and sometimes of a dull yellow colour; the skin is slightly reddened, and the tongue slightly furred; there is a feeling of uneasiness and obtuse pain at the epigastrium, not increased on pressure; sighing, oppression, nausea, and vomiting of mucous or bilioso-mucous matter; costiveness; urine abundant, crude, and pale. The pulse is easily compressed, seldom accelerated; often slow. There is no thirst; breathing is sometimes accelerated, more frequently natural. On the second or third day, a miliary eruption breaks out over the body, with conical pimples filled with a transparent fluid, and surrounded by a red areola. Sometimes, there exist only red or rose-coloured spots, or larger pustules. The disease runs its course in from five to seven days, when vesication of the eruption and desquamation of epidermis commences.

This conclusion will not be neutralized by the fact that the sweating miliary fever has occasionally prevailed in hot latitudes, as at Gizeh, on the banks of the Nile,¹ for it first made its appearance in Germany, and is endemic, or more generally breaks out and prevails in cold and humid localities, and under circumstances unfavourable to the production of the yellow fever.

Relapsing Fever.—The peculiar form of continued fever observed in several parts of Scotland—Edinburgh, Leith, Glasgow, and Dundee—in 1817, 1843, and 1847; in Dublin in 1731, 1741, 1800, 1801, 1816, 1817, 1818, and 1826; in London in 1847, as well as in Germany at various epochs, and which, from a peculiarity in its course, has received, from some writers, the name of relapsing fever, presents, on some points, a resemblance to the yellow fever—with which, indeed, it has by some been confounded. It may not be improper, therefore, to inquire into the correctness of this view, and to see how far the parallel between the two diseases will hold.²

¹ Desgenette, *Hist. Méd. de l'Armée D'Orient*, p. 212.

² Cormack, *Natural Hist., Path., and Treat. of the Epid. Fever at present prevailing in Edinb. and other Towns*, pp. 182; Alison, *Remarks on the present Epidemic Fever, Scottish and N. of Engl. Med. Gaz.*, i. 1–4; Henderson, *On some of the Characters which distinguish the Fever at present epidemic from Typhus Fever*, *Edinb. Med. and Surg. Journ.*, lxi. 201; David Smith, *Some Account of the Epidemic Fever prevailing in Glasgow*, *Edinb. Journ.*, lxi. 67, lxii. 62; Craigie, *Notice of a Febrile Disease which has prevailed at Edinburgh during the Summer of 1843*, *Edinb. Journ.*, lx. 411; Mackenzie, *Some Account of the Epidemic Remittent Fever prevalent in Glasgow in 1843*, *Lond. Med. Gaz.*, Nov. 24, 1843; Arrott, *Letter on the present Epidemic of Dundee*, *Scottish and N. of Engl. Med. Gaz.*, i. 129; Jenner, *On the Identity or Non-Identity of Typhoid and Typhus Fever*, *Monthly Journal of the Medical Sciences*, 1850, pp. 102; Jenner, *On the Identity or Non-Identity of the Specific Cause of Typhoid, Typhus, and Relapsing Fever*, *Med. Chir. Trans.*, xxxiii. 26; Jenner, *Typhus Fever, Typhoid Fever, Relapsing Fever, and Febricula, the Diseases commonly confounded under the term Continued Fever*, illustrated by Cases collected at the Bedside, *Medical Times*, 1849–'51; John Charles

In the relapsing fever, we note suddenness of attack, severity of articular pains, high fever, pain in the head, pain and tenderness about the epigastrium, and vomiting. Delirium is unfrequent, but restlessness and sleeplessness are great; the skin is hot, the temperature rising sometimes several degrees above 100; the tongue is white; the thirst is sometimes considerable; the pulse is very frequent—seldom below 100, and varying from that to 120, and even higher. In a certain number of cases, on the third or fourth day, the skin, particularly on the face, assumes a slight bronze tint. This is followed, on the fifth or sixth day of the disease, by an attack of jaundice, attended with severe vomiting—the matter ejected being bilious, bright grass green, sometimes coffee-ground like and absolutely black. A day or two after this, when the symptoms appear to become more alarming, there ensues in most, though not in all cases, a most remarkable series of symptoms, followed by a complete intermission of all symptoms, and an apparent restoration to health. This change, which has received the name of “crisis,” is attended with, or preceded by, a profuse sweat, or a discharge of some other kind, as diarrhœa, epistaxis, diuresis, or menorrhagia. The crisis, though almost always present, does not invariably occur, as some patients, about the time it should take place, begin to improve, without any notable discharge marking a sudden transition from one state to another.

In this brief enumeration of the distinctive phenomena of the relapsing fever, we certainly discover some of those of the yellow fever. But, on more careful examination, we shall find that, even on the points mentioned, the analogy is but remote, certainly not as close as might at first sight appear; while on many others the difference is sufficiently marked to justify the conclusion that the two diseases belong to distinct categories of febrile complaints. It is scarcely necessary to remark that the early symptoms—muscular and articular pains, considerable feverishness, headache, great tenderness about the epigastrium, vomiting, restlessness, sleeplessness, white tongue, thirst, quick pulse, &c.—cannot serve to establish an identity of nature or close correlation between the disease in question and the yellow fever. Many of them are common to other febrile diseases of diversified character, and cannot, therefore, be appealed to for the purpose in question; and from the fact that a

Steale, *Pathological Statistics of Glasgow Infirmary for 1847*, Edinb. Med. and Surg. Journ., lxx. 145; Paterson, *Statistics of the Barony Parish Fever Hospital of Glasgow, in 1847-'8*, Edinb. Med. and Surg. Journ., lxx. 357; Robert Paterson, *An Account of the Epidemic Fever of 1847-'8 in Edinburgh*, Edinb. Med. and Surg. Journ., lxx. 371; Orr, *Hist. and Stat. Sketch of the Progress of Epidemic Fever in Glasgow, during the Year 1847*, Edinb. Med. and Surg. Journ., lxx. 363; Robertson, *Notes on the Epidemic Fever of 1847-'8*, *Monthly Journal*, 1849, p. 368; Baker and Cheyne, *Account of the Rise, Progress, and Decline of Epidemic Fever in Ireland*, London, 1821; West, *On the Efficacy of Bloodletting in the present Epidemic Fever of Edinburgh*, 1819; J. O'Brien, *Report of the House of Recovery and Fever Hospital of Dublin, for the Year ending 1827*; *Trans. of the Assoc. of Fellows and Licentiates of the King and Queen's College of Physicians in Ireland*, v. 266, 512 (1828); Ruttty (John), *Chronological History of the Weather and Seasons, and of the prevailing Diseases of Dublin*, pp. 75, 90, 130, 303-4, 319—London, 1770; Christison, *Library of Medicine*, vol. i.

few are more frequently noticed in these two forms than in the others alluded to, we can only infer the existence of an unimportant coincidence, the phenomena being of an insufficient character to be held as pathognomonic.

But, even in reference to some of these symptoms, we shall discover, on closer examination, that they present a different aspect in the two diseases, while others noticed in the yellow fever are not observed in the relapsing fever. We find in the description of the relapsing fever no mention of the torturing lumbar pain which forms a marked characteristic of the yellow fever, of the peculiar frontal or orbital pain, &c. In the former, also, the pains in the head, back, and limbs do not always disappear or subside during the remission. In some cases, they do not abate, or they are actually aggravated; "or, the original febrile pains subsiding, are followed by pains which present all the characters of rheumatism or rheumatic gout" (*Craigie*, p. 412). Not so in yellow fever. Although, in the latter, high febrile heat and increased energy of the circulation are frequently observed, they are not as universally so as in the other—being moderate, and even often absent in the more severe cases. The peculiar bright, fiery, and suffused eye, and the excessive redness of the face, which play such prominent parts in the symptomatology of the yellow fever, have not been observed in the other disease. In the yellow fever, the tenderness over the stomach is principally limited to the epigastric region; sometimes it extends to the right side. In the relapsing fever, though often felt at the epigastrium, it is frequently more severe over the region of the spleen, and seems connected with a morbid condition of that organ. To this it may be added that in the relapsing fever the symptoms of reaction are at their height on the third or fourth day—a period when they have, in the very large majority of cases, disappeared in the yellow fever. The febrile paroxysm in the former continues from five to nine days, more frequently from five to six days. In the yellow fever, except in very mild cases, it seldom extends beyond the third, and is often much shorter. True it is, that Dr. Craigie (p. 416) and other writers have noted the mildness of the symptoms about the third day—the headache then abating, the thirst diminishing, and the patient expressing himself free from complaint. But the pulse is, nevertheless, rarely much reduced; on the fourth day, the symptoms are worse, and a decided amelioration only occurs on the fifth, or later. This is not the course in the yellow fever. The period of cessation, which, as must be foreseen from what has just been stated, takes place at different times in the two diseases, is less attended with critical discharges in the yellow than in the relapsing fever.

The relapsing fever is characterized, in a great number of cases at least, by an eruption of small spots, round, purple, unaltered by pressure, and closely resembling flea-bites. In the yellow fever, this eruption, though sometimes noticed, is not sufficiently frequent to be regarded as characteristic of the disease. The yellowness of the surface, which is so generally seen in the yellow fever, during life or after death, is an unfrequent symptom in the relapsing fever. In 1817, Dr. Welsh, who terms it a trifling symptom, noted it only once in about thirty cases (p. 73). Dr. Douglass saw it twenty-nine

times in two hundred and twenty cases. Dr. Jackson saw it in thirty-one out of three hundred cases. In Silesia, it was comparatively uncommon. In London, on the contrary, Jenner considers that it occurred in nearly one-fourth of his cases.¹ But, were it even more frequent in the relapsing fever, than we find it to be, its occurrence would not be sufficient, unconnected, as it is, with other distinctive symptoms, to prove the identity of that disease with yellow fever, in which, as I have already stated, the symptom cannot be viewed as pathognomonic, failing, as it does, to occur in many cases, and appearing as frequently, if not more so, in other febrile diseases but remotely allied to the former.

Still less can we discover, in the nature of the matters ejected from the stomach, an evidence of identity or close analogy between the two diseases. The bright grass-green matter noticed in the relapsing fever is seldom observed in cases of pure yellow fever, more especially on the fifth or sixth day. The dark-green, grumous-looking vomit, noticed in the former, when mixed with water, gives to this a greenish tint; while the real black coffee-ground matter so usually thrown up in the latter disease, or found in the stomach after death, is but seldom seen in the other disease (*Smith*, lxii. 67)—scarcely more frequently than in other diseases of an avowedly dissimilar character; and while it may be viewed as an almost sure indication of approaching death in yellow fever, it is far from being so in the relapsing, as shown by the results obtained, especially by Dr. Kilgour, at Dundee, where such cases were very frequent, and yet the mortality very small. The jaundice of the relapsing fever is accompanied with free secretion of bile, generally with tenderness over the liver, which is sometimes enlarged—phenomena which seldom, if ever, occur in the yellow fever. The mortality differs widely in the two diseases. In uncomplicated cases of the relapsing fever, few, if any, die. In fatal cases, death seems to result from thoracic or abdominal complications—pleurisy, pneumonia, and dysentery. The fatal cases in 1843 averaged from two to six per cent. In 1847, they amounted, at Glasgow, to 6.38 per cent., and at Edinburgh, to 3.14 per cent. In 1843, Douglas lost nineteen in two hundred and twenty cases, or 8.63 per cent. Kilgour, at Aberdeen, says it was under one in thirty. Mackenzie, at Glasgow, $3\frac{1}{2}$ per cent. Craigie, one in sixty. Alison, one in thirty. To those acquainted with the yellow fever, it is not necessary to state that such a rate of mortality has never been heard of, and will probably never occur, except in lucky hands, such as those already referred to; while death in that disease is usually the result of the morbid cause fulfilling its office of destruction, without the necessary aid of extraneous complications.

But much more conspicuously do the fevers differ in regard to the phenomena which succeed to the crisis. In the European fever under examination, the crisis is followed, in most cases, by a period of perfect intermission; in some cases by a remission only. In most instances, this state of recovery or amendment is suddenly succeeded, after a few days, by a second accession of

¹ *Med. Times*, Dec. 1850, p. 646; *Brit. and For. Rev.*, July, 1851, p. 9.

fever—the so-called relapse. It occurs, on an average, on the fourteenth or fifteenth day—the extremes being the ninth and forty-eighth day. The symptoms during the second accession are similar, except it may be in point of severity, to those observed in the first. They continue three or four days; are followed on the eighteenth or twentieth day of the disease by a second crisis. This is followed by another intermission which, in its turn, is not uncommonly succeeded by a second relapse; after which, comes a third intermission, then another, &c.

In the yellow fever, on the contrary, the occurrence of a relapse is rare. The period of calm, which succeeds to a febrile paroxysm of from one to three days, is, as has been shown, followed, in the majority of cases, by severe and dangerous symptoms; but not by fever—certainly by nothing resembling the phenomena of the period of excitement; and while in fatal cases death occurs without the accession, from the decline of the first stage to the last moment of life, of any sign of reaction, in those that recover, the symptoms of the second and third stage, which are not characterized by phenomena of excitement, gradually decline; convalescence is re-established, and recovery, if not retarded by improper treatment, or some complication, soon follows.

The relapsing and yellow fevers do not prevail at the same season of the year and under similar atmospherical conditions. True it is, as appears from the accounts that have been handed down to us, that the former fever has, on several occasions, occurred during the summer and become more general as the heat increased. But while such was undoubtedly the fact, it is not the less certain that the disease, though uninterrupted or even aggravated by heat, has often broken out and spread at a season of the year when cold must have been still very considerable. The first cases noticed at Glasgow in 1843, occurred in January;¹ though, according to Dr. Mackenzie,² who cites Dr. Kennedy, cases had presented themselves in September of the preceding year. In Edinburgh (1843), the disease began to prevail in February,³ or at the close of March or beginning of April, according to other authority.⁴ But, whether the outbreak occurs in January, February, or March, those acquainted with the temperature of Glasgow and Edinburgh in those months, will easily perceive that if the relapsing fever has manifested itself then and there, a high degree of temperature is not indispensable to its production. It is needless to remark, that the reverse takes place in regard to the yellow fever; for if there is one point, connected with the etiology of that disease, upon which no difference of opinion can be pointed out, it is the absolute necessity of a high and continuous range of thermometrical heat to call into action the cause to which it owes its existence.

In both diseases the blood is found diseased. Dr. Cormack, who calls attention to this in relation to the relapsing fever, states that the blood formed a homogeneous mass. To this morbid state of the fluid he refers the ecchymoses formed around flea-bites and other slight injuries of the skin,

¹ Smith, Edinb. Journ., lxii. 64.

² Henderson, pp. 61, 216.

² Med. Gaz., Oct., xxxii. 225. 1843.

⁴ Craigie, pp. 60, 414.

purpura, hemorrhages, &c. When examined under the microscope, whether it was taken in the pyrexial or apyrexial state of the disorder, the blood was discovered to contain an unusual number of pus-globules, and in some cases all the globules were found serrated and notched. In much of this, the disease approximates to the yellow fever, in which the blood is found altered, and gives rise, from its diseased condition, to the symptoms mentioned. But these appearances in the yellow fever are found, principally, during the latter or apyrexial stage; and besides, being common to several forms of fever, as well as to cholera and other zymotic diseases but slightly allied to the latter, their occurrence in the two diseases before us, cannot be urged in proof of the identity of these.

Nor can we appeal for the purpose of corroborating that opinion to the fact, that in the relapsing, as well as in the yellow fever, the urine is often suppressed, and that this symptom almost invariably portends a fatal issue. The same phenomenon and results are observed, though, perhaps, not so frequently, in other diseases.

While, from some of the preceding phenomena, no proof of the identity in question can be adduced, and while many others disprove it, the anatomical characters of the relapsing fever bear but a very slight analogy to those of yellow fever. In the former, the spleen is often enlarged—in some cases, so much so, as to be easily distinguished projecting beyond the lower and anterior margin of the hypochondrium. Dr. Henderson supposes that the organ—whose enlargement could be detected during life by the feel, or percussion—must have increased to four or five times its ordinary size. Nothing of the kind is discovered in yellow fever, during life or on dissection; while, in the published accounts of relapsing fever, we find nothing to justify the belief that the liver and other parts exhibit the changes noticed in the yellow fever.

With all these facts before us, we can find no reason to refuse coinciding with Dr. Craigie when he expresses the opinion that “it is scarcely possible, with any consistency in pathology, or common observation, to admit even the resemblance between the two diseases in question” (p. 416).

CHAPTER XXVIII.

DIAGNOSIS CONTINUED.

Bilious Remittent Fever.—Much more frequently than with any of the preceding diseases, the yellow fever has been confounded with the gastric and bilious remittent fevers of tropical climates and of the summer and autumnal months of cooler latitudes. So numerous, indeed, are the points of contrast they present in some cases, that, without close attention, the diagnosis is not always easily established, and many pathologists refuse to

acknowledge the existence of any specific difference between them, maintaining that all summer and autumnal fevers, from the slight intermittent or remittent to the deadly yellow fever, are identical in nature, arise from the operation of the same cause, and vary only in respect to the degree of their violence and malignancy. The idea of the close alliance or identity of all such fevers—of regarding them simply as varieties of one and the same disease—is of old origin, and has been supported by high professional authorities. In regard to the fevers of the West Indies, the opinion was early advocated by Towne (p. 20), Chevalier (p. 10), Rouppe (p. 304), Bourgeois (p. 416). Dr. Lind states, that having considered this disease with attention, he is of opinion “that the remarkable dissolution of the blood, the violent hemorrhages, the black vomit, and the other symptoms which characterize the yellow fever, are only accidental circumstances in the common fever of the West Indies. They are to be esteemed merely as adventitious, in the same manner as purple spots and bloody urine are in the smallpox, or as a hiccup in the dysentery; like these, they only appear when the disease is accompanied with a high degree of malignity, and therefore always indicate great danger” (p. 133, 6th ed.).

Dr. R. Jackson, in his early treatise on the diseases of Jamaica, regarded the yellow fever as distinct from the remittent. In his *Outline of Fever*, published in 1798, he relinquished this opinion, attributing the change to a more enlarged experience, and more accurate observation; and affirming that “the disease is actually one—the action of the cause modified by circumstances of the subject.” (*Advertisement*, p. 6.) The same views were entertained by Dr. Rush and his school. It was warmly espoused by Dr. Deveze (pp. 196, 199, 200), and ably advocated by Dr. Bancroft (p. 291), Hillary (p. 143, &c.), Ferguson (*Med.-Ch. Tr.*, viii. 152), Musgrave (*Ib.*, ix. 123), Chervin (*Réponse à M. Guyon*, p. 214; *Report on Rufz*, pp. 64, 71, 95), and others too numerous to be mentioned here.¹

¹ S. Brown, p. 1; Potter, p. 2; Drysdale, i. 22; E. H. Smith, pp. 107, 144; Valentin, pp. 22, 39, 57; *Ib.*, Journal Univ., xi. 143; *Ib.*, *Traité de la Pleurésie*, p. 148; Ramsay, *Med. Repos.*, viii. 365; Davidge, p. 71; *Ib.*, *Repos.*, ii. 83; Pascalis, *Fev. of 1797*, p. 111; Waring, pp. 4, 5, 8; Merrill, ix. 242; Jameson, *Recorder*, vi. 435; Griffith, *Recorder*, xiv. 240; Hill, v. 90; Thomas, p. 59 (1st ed.), p. 33 (2d ed.); De Rosset, *Repos.*, ii. 143-4; Vaughan, p. 17; Daniel, p. 35; Huestis, *N. Y. J.*, iv. 397; *Ib.*, *Dis. of Alabama*, p. 109; *N. O.* 1839, p. 316; Caldwell, *Med. Mem.* (1801), p. 208; *Ib.*, *Feb. Miasma*, pp. 477, 488; Bayley (1795), p. 55; Rouppe, p. 302; Pinkard, ii. 468; J. Hunter, p. 62; T. Clark, p. 1, &c.; H. McLean, p. 71; Rufz, p. 59; Ralph, ii. 54; Caillot, p. 65; Béguerie, pp. 21, 88; Catel, p. 7; Gilbert, p. 77; Wallace, xlv. 271; Arnold, p. 33; Bertulus, p. 7; Proudfoot, p. xxvii. 246; Gillkrest, ii. 265; Frazer, *Med.-Ch. Rev.*, xiii. 338; Amiel, in Johnson, p. 265; T. Smith, *Edinb. J.*, xxxv. 42; Lassis, p. 57; Robert, ii. 25, 29, &c.; Burnet, *Rept. on the Bann*, p. 48; *Ib.*, *Fev. of Mediterranean*, p. 252; *Ib.*, *Correspond. about the Eclair*, p. 78; Donnet, in Burnett, p. 482; Doughty, p. 189; R. Jackson (*Spain*), pp. 6, 53; Bryson, pp. 77, 250; Tommasini, i. 46; McWilliams, *Rept.*, pp. 77, 105; Jameson, *Dublin Quart. Med. J.*, xvi. 345; *Med.-Ch. Rev.*, 1848, i. 300; Bone, p. 7; Pennell, *A Short Report upon Yellow Fever as it appeared in Brazil in 1849-1850*, p. 23, Rio Janeiro, 1850.

The grounds on which this opinion is founded have been summed up by several of its advocates: 1. The yellow fever, when it prevails epidemically in temperate climates, does so in summer and autumn, the very seasons in which intermittent and remittent diseases are most rife. 2. It shows itself only in those localities where the latter diseases originate and prevail. 3. When, in equinoctial regions, the yellow fever spreads among the unacclimatized, periodical fevers usually reign among the natives and long residents. 4. The meteorological phenomena which exercise a marked influence on the progress of the yellow fever, exercise an analogous influence on that of periodical fevers. In both instances the course of the disease, as an epidemic, is arrested by frost, or, as occurs in tropical climates, by a change of wind, heavy rains, or excessive drought. 5. It is well known that the effluvia which give rise to the latter are wafted by the wind. The same thing occurs, though to a more limited extent, in relation to those that produce the yellow fever. 6. Experience has shown, that in cities where the yellow fever prevails, the disease is more rife in low and ill ventilated localities, small and narrow streets, the ground-floor of houses, and the like. The same remark may be made in regard to periodical fevers, which are also found to be more prevalent in similar places. 7. It is a well-known fact, that the exhalations which produce periodic fevers are greatly more active during the night than the day. Those that give rise to the yellow fever, in like manner possess much greater power after sunset than before. 8. In equinoctial regions, the yellow fever generally reigns almost exclusively over the unacclimated; so, also, do intermittent and remittent fevers, which attack, in preference, individuals removing from salubrious to marshy localities, as has been abundantly proved during the successive occupations of Italy, Spain, the Ionian Islands, the Morea, and Algiers, by the English or French troops, and in many other places on both continents. 9. Individuals who have taken an intermittent fever in a marshy situation, moderate the effect of the poison and hasten their recovery by removing to a healthy place; the same result is obtained in reference to the yellow fever, though in a less degree, owing to the greater rapidity with which the paludal intoxication has taken place, and the greater extent to which it has been carried. 10. The symptoms supposed to characterize the yellow fever—the aspect of the eyes, the nature and seat of the pain in the head, the coloration of the face and surface, the expression of the countenance, the morbid state of the stomach, the nature of the matters ejected from that organ or by stool, &c., are observed in the other form of disease, and can, therefore, establish no difference between them. 11. The yellow, like the other forms of autumnal fever, assumes, at various times, the remittent and the intermittent as well as the continued type, and in that respect, therefore, differs in nothing from the latter. 12. They are all convertible diseases. Cases commencing with symptoms regarded as characteristic of the yellow fever frequently assume, from the effect of treatment or other circumstances, the garb of a common remittent or intermittent; and, on the other hand, cases which, at the onset, present the symptoms of a remittent or intermittent, will often acquire the characters of the yellow fever. 13. Doubt-

less, if we compare a mild case of remittent fever with a severe case of yellow fever, a considerable difference will be noticed between the two; but if the comparison be made between a severe case of remittent and a mild one of yellow fever, this difference disappears. In fact, a continued chain can usually be traced, link by link, from the simple and mild periodic fever up to the most concentrated form which invades the unprotected in tropical regions and elsewhere during severe epidemics. 15. The morbid appearances revealed on dissection are the same in remittent as in the yellow fevers. 16. Finally, neither the yellow fever nor the other forms of febrile complaints mentioned are propagated by contagion.

Such are the principal reasons assigned by the advocates of the unity of the diseases under consideration. But the theory they have propounded on the subject, plausible as it may appear on a hasty glance, and numerous and ably supported as it undoubtedly has been, and continues to be, is open to serious and, indeed, insuperable objections, and has hence met with strong opposition from a large number of medical writers, contagionists as well as non-contagionists.¹

By these, facts and arguments have been accumulated to disprove the doctrine of unity as applied to the diseases in question, and to show that, so far, its advocates have done little beyond pointing out a fact no one has denied, namely, the existence of an affinity, or family likeness, in point of symptomatology, pathology, and etiology—between the two diseases; while reasons, stronger than any they have adduced, may be found to establish the independence of each of these and their claims to be considered in the light of different and distinct complaints, and consequently to occupy a separate position in every nosological arrangement. Such are the views which a prolonged and attentive examination of the subject has led me to entertain on a subject, the importance of which, in a pathological, therapeutical, and prophylactic point of view will not be denied. It ought to be stated before

¹ Warren, p. 2; Moseley, pp. 138, 203, 411; Lempriere, ii. 66, 80; Desportes, i. 230; Stevens, p. 193; Rochoux, xi. 378; Gillespie, p. 59; Pym, i. 223; Gilpin, v. 315, 321; Chisholm, i. 239; Savarésy, p. 251; Blane, p. 425; Osgood, p. 27; Madrid, p. 15; Imray, liii. 79; Ibid., lxiv. 333; Dariste, p. 111; Diekinson, *Introd.*, pp. 1, 2; Veitch, p. 18; Bally, p. 3; Currie (1793), pp. 39, 64; Ibid., *Bil. Fev.*, p. 207; Barnwell, p. 366; Hosaek, *Essays*, iii. 429; Lining, i. 406; Monges, ii. 56; J. Wilson, pp. 98, 175; Blair, p. 22; Chapman, *Med. and Phys. Journ.*, ix. 129; E. H. Smith, p. 109; Tully, p. 294; Townsend, p. 179; Beck, *N. Y. Med. and Phys. Journ.*, iv. 401; A. Smith, *Trans. N. Y. Acad. of Med.*, i. 51; Klapp, *Med. Ree.*, iv. 86; Parrish, v. 21; Simons, pp. 20, 21; Diekson, pp. 336, 37; Ibid., *Med. and Phys. Journ.*, iii. 265; Wood, i. 303; Jolivet, p. 12; Seamen, p. 11; Girardin, p. 44; Dalmas, p. 11; McCraven, *Tr. of Med. Assoe.*, v. 670; Irvine, p. 18; Chabert, p. 138; *N. O.*, 1839, p. 336; Kelly, *Am. Journ.*, *N. S.*, xiv. 375; J. M. Smith, on *Epidemics*, p. 109; Bowen, *Med. and Philos. Register*, iv. 449; Noreom, *ibid.*, i. 17; Seagrave, *ibid.*, iii. 417; Copland, iii. 144, 147; Peixotto, i. 413, 414; Fellowes, p. 22; Berthe, p. 137; Peter Wilson, p. 72; Stone, vi. 549; Barry, in Boyle, p. 27; Nott, *Charleston Journ.*, iii. 2; Ibid., *N. O. Journ.*, iv. 570; Lewis, *N. O. J.*, iv. 13, 151, 166; Lallemant, *Fev. of Rio Janeiro, Report of N. O. Com.* (1853), p. 163.

proceeding further, that while doubting or denying the identity of yellow fever with the bilious remittent of this and other countries, I wish to include under the former name the several varieties and grades of the disease which is almost universally recognized as such in temperate and tropical regions, and thereby avoid the error committed by Dr. Copland, who draws a line of demarcation between true yellow fever, or, as he denominates the disease, the hæmagastric pestilence, and a form of fever of tropical climates which he describes under another name—the malignant remittent—but which, on strict analysis, is found to differ in nothing from the former. Dr. Copland, let it be stated, accuses Dr. Bancroft of misleading the inexperienced, and long mystifying many (iii. 146). This may be granted. But, at the same time, it may be doubted whether Dr. C. has not been guilty of the same offence, though in another way; for, while Bancroft has made of *two* diseases *one*, the other has made of *one* disease several—not without admitting, however, that what he calls the malignant remittent, assumes sometimes the type and the symptoms of the true yellow fever (iii. 140), but that, nevertheless, it differs materially from it in the fact that it has not yet acquired the contagious property!

But, let this be as it may, it is to be borne in mind, that several of the circumstances enumerated in proof of the exact similarity, in point of nature, between the yellow and ordinary autumnal fevers—the appearance of all at the same period of the year—their prevailing in the same localities, and among the same classes of individuals—their being under the influence of the same meteorological conditions—favoured in their origin by heat and humidity, and arrested by cold, &c., their prevailing most in low, ill-ventilated places and close apartments—their being more apt to attack at night—their effects being mitigated by a removal to a more healthy situation—their efficient causes being wafted by the wind—all these circumstances, I say, prove, doubtless, that these diseases are all the products of morbid agencies or poisons of kindred nature; but from this it does not necessarily follow, that they are identically the same, and differ only in respect to the degree of their violence; to the extent to which the intoxication has been carried, and to the greater concentration of the poison. Nothing can prevent the belief, that malarial exhalations may differ so materially in their composition as to give rise to disease which, while approximating in this, that they are malarial and members of the same family, differ materially in every other respect. The question then to be decided would be, whether or not such a difference exists in the nature of the poisonous agents producing the fevers in question. Until the question is satisfactorily settled in the negative, it will be unsafe to affirm that the complaints they occasion, however dissimilar in many points, are identically the same. I shall, in a future chapter, adduce some facts calculated to prove the reality of this difference, and therefore will recur to the subject here, only so far as is necessary for the elucidation of the question more particularly under consideration.

Whatever be the nature of the morbid agencies giving rise to those diseases, and however allied they may be to each other, there are facts sufficient

to show that the laws of their progression and diffusion are not exactly the same. The cause of autumnal fevers is more under the influence of the wind than that of the other. It is wafted to considerable distances, and ascends in the same way to great heights. Numerous facts in illustration of this have been collected by the author of the present work, and published elsewhere.¹ The horizontal and altitudinal ranges of the cause of yellow fever are much less considerable; indeed, may almost be said to be null; the area within which it exercises its morbid influence, or is carried, being comparatively very circumscribed, and the height to which it ascends being equally so. It creeps, as it were, along the surface of the earth, and, in general, proves innocuous to those who reside at a small elevation above the infected district, or occupy the upper apartments of houses situated in the very focus of the infection. The cause of intermittent fever is not readily stopped in its migration by water—being carried to great distances at sea, or across lakes and rivers. In respect to the efficient cause of yellow fever such is not the case; for ships at anchor, at even a comparatively limited distance from a sickly port, remain free from the disease, so long as the men on board abstain from going ashore.²

The prevalence of both forms of fever in hot latitudes and in hot weather, cannot be adduced in proof of their identity; for, on the same ground, we should be led to admit, what has never been admitted, that other diseases, which generally appear under like circumstances—cholera, dysentery, diarrhoea, and hepatitis, to say nothing of scarlatina, measles, influenza, whooping-cough, &c., are also identical with the yellow fever. If the cause of both diseases were the same, they ought to exist in the same places, and where bilious remittents prevail most frequently, the yellow fever should prevail likewise. Such, as already seen, is the position assumed by the advocates of the identity in question. But experience shows that these statements, as well as many others, to which attention will be called as we proceed, are greatly at variance with well established facts. Heat is certainly necessary for the development of both; but the degree of it required for the elimination and continued spread of the causes producing them, is not the same in each. The yellow fever, as will be pointed out more particularly in a future chapter, never or seldom breaks out except after a more or less prolonged continuance of very hot weather. It may, and often does, continue to prevail after the temperature has considerably lowered; but it never originates except under the circumstances mentioned. The other fever continually breaks out at a temperature too low for the production of the yellow fever. A temperature of 60° is necessary for its manifestation, and it never prevails as an epidemic where the temperature of the summer falls below 65°. On this side of the Atlantic, the latter fever prevails from the tropic to the latitude of 35°, or a little higher, and has occurred only once above the latitude of 32°. The other prevails from the tropics to the latitude of 44°, and is often epidemic and mortal as far north as 43°. (*Drake*, ii. 354.) But, experience shows that

¹ Pneumonia, &c., pp. 210, 220.

² *Ibid.*, pp. 130, 217.

though heat is necessary to both, their localities also are very different—that where the one is widely diffused, the other is often never or seldom encountered. Thus, as already stated, the bilious fever abounds—even in a malignant and fatal form, but with features very unlike the yellow fever—in places where the latter is never seen.

In the islands of Dominica, Demerara, and St. Lucia, common miasmatic fevers prevail annually, while the yellow fever appears at irregular intervals. In Barbadoes, on the contrary, the latter disease is of frequent occurrence, and the former scarcely known. Bilious remittent fever, in its worst forms, is as prevalent in India, in Eastern Europe, and in all parts of Africa, as in our southern States, the West Indies, and the western coast of Africa; nevertheless, the yellow fever is seldom if ever seen there; while it is very common in the others. Bilious remittents and intermittents prevail as extensively, and with as much violence, in the interior of unhealthy countries as near the sea; the yellow fever, on the contrary, is confined within narrow limits, and is generally observed on the sea-coast, and along navigable streams, and seldom extends far into the interior. The yellow fever, as we shall see, has often been known to arise from the foul exhalations of ships; no instance, so far as I have been able to ascertain, has as yet occurred of epidemics of remittent or intermittent fevers having broken out at sea, or been traced to the decayed timber, or dirty bilge-water, or fermenting cargo of a ship. If cases appear there, they have invariably been brought from elsewhere. The yellow fever is, strictly speaking, a disease of cities, or of places containing a dense population. That it has sometimes attacked small towns, villages, plantations, or rural districts, is true; but such instances are rare, and have generally occurred along large watercourses; and it should be borne in mind that plantations are virtually small towns, where the population is concentrated within a narrow compass, and often amounts to one or several hundred souls. In some of the cities or towns thus affected, the disease spreads to a greater or less extent all over; but, in general, it prevails within a limited area, and often in spots where the remittent fever does not penetrate. Take Philadelphia as an example.

In former days, when the city was of limited extent, with few improvements; with buildings scattered about, and leaving open and unimproved spaces between them; with a marshy stream running through the greater part of it; with ponds, natural and artificial, spotted over the plot in various directions, and with unpaved streets—fever was of common occurrence, and epidemics were not unfrequent. At present, common malarial fevers are unknown in the city proper, as well as in the compactly built and well-drained portions of the suburban districts. If we wish to meet with them, we must go to the outskirts of these districts, or to some distance from them, to the open meadow ground of the neck, or to other unimproved surfaces of the vicinity; but, more particularly, to the marshes which still exist along the river banks.

Our townsman, Dr. Emerson, who has devoted considerable attention to the subject of public hygiene, and furnished several excellent essays on the vital statistics of Philadelphia, has conclusively shown that the influence of

the sickly air is expended upon the comparatively limited portion of the population living in the environs and outskirts of the city. During the periods embraced in his calculations—and the same holds good in all other times and places—the fever, in some of its forms, was almost universal; whilst in the more dense and well-paved parts the air seemed unusually healthy, and where remittents and intermittents were met with, they could almost invariably be traced to exposure to night air in the country or suburbs. Never, he remarks, was a stronger demonstration afforded of the resistance made by cities to the influence of country malaria, than our late experience has furnished. Great as was the amount of sickness during the epidemic of 1822 and 1823, it was confined almost entirely to the comparatively small proportion of population inhabiting the unpaved or ill-paved environs. “Our observation,” Dr. E. adds, “on this and other occasions, has led us to ascribe this exemption, for the most part, to the pavements, which, by effecting a perfect drainage, prevents exhalation, at the same time that it admits of the total removal of vegetable and animal matters, the sources of foul and unhealthy emanations. The chief motive for paving the streets and sidewalks is usually convenience; but it has always appeared to us that by far the most important object achieved by it was the preservation of health.” Since the time to which this has reference, thirty years have elapsed. In the interval, the compact, dense, and well-paved parts have extended far beyond their boundaries at that time, and, with their expansion, the disease has receded. While such is the case with respect to autumnal fevers, the yellow fever, when it breaks out among us, is invariably limited to a small area along the wharves, and in the adjacent streets, where we never hear of a case of the former originating.

Bilious remittents or intermittents, though occasionally seen in city localities, prevail more generally in the suburbs or adjacent country, to which the other never extends. Although it often happens that an epidemic of yellow fever is accompanied with a like prevalence of remittents and intermittents in the surrounding country, or, perchance, in the infected place itself, the occurrence is not constant; for instances are found when, during severe visitations, the country or suburbs have remained free from common malarial fevers. On the other hand, in some very unhealthy seasons, both in the West Indies and in our Southern and Middle States, when the fevers have prevailed most extensively, the yellow fever has not made its appearance; and yet, in the former instances, there existed very mild cases which, notwithstanding the absence of malignant symptoms, differed widely from the remittent fever; while in the latter the remittent was often violent, and proved fatal, without, however, bearing any resemblance to the yellow fever. Besides, were coexistence of the two diseases always observed, it would not prove identity; but only the existence of a powerful epidemic constitution of atmosphere, together with the concurrence of the two sets of local causes producing both diseases, and the diffusion of which is promoted by the former.

The circumstance of yellow fever attacking the unprotected, while paludal fevers—bilious remittent and intermittent—affect the acclimated or

protected, is no proof of the identity contended for, inasmuch as it is as reasonable to suppose that the two diseases coexist, and that while the former class of individuals are exposed to both, but suffer too readily from the severer form to have the milder, the acclimated, who are proof against the former, suffer from the other. From this coexistence of the two diseases we may derive an explanation of the bilious preceding or following an epidemic visitation of the yellow fever, or existing during its continuance, but we cannot appeal to these circumstances in proof of the identity in question; for the yellow fever may be supposed to break out after the other form, to continue with it, and, ceasing before it, to leave it to follow its own course. Nativity or long residence in countries subject to bilious remittents is of no or little avail against those diseases in relation to which there is only an imperfect acclimatization. There is no place where a contrary result has been observed, whether in the miasmatic portions of this country, from Texas to Maine, the Campagna and the Pontine Marshes of Rome, the Maremma of Tuscany or Lucca, the Bresse in France, the jungles of India, the swamps of Walcheren, &c. In all these places, strangers are doubtless more prone to the disease than residents; but these are often affected, too, and in a larger proportion than is the case with regard to the yellow fever anywhere. But, whatever may be the case in relation to this matter, individuals who have become inured to the atmosphere of localities subject to autumnal fevers are, so far from being protected against the yellow fever, just as liable to be attacked with this disease, when they visit an infected district, as though they came from salubrious regions. Reversely, persons acclimatized to the atmosphere of yellow fever cities are open to the inroads of autumnal fever on removing to malarial districts. There is no reciprocity on this point.

An attack of the ordinary form of bilious remittent affords no protection against a second—a mere repetition of the disease—the same disease being of frequent occurrence, and the liability of the system being indeed increased by previous attacks. We shall have occasion to see that the reverse is the case in the yellow fever, from which the acclimatized seldom or never suffer, and in which second attacks are seldom seen. While this is the case, an attack of bilious fever, however severe, does not protect against the yellow fever, and *vice versâ*.¹

We have seen that relapses, properly speaking, though occasionally encountered, are not of frequent occurrence in the yellow fever. When they do occur, we do not find that the symptoms exhibited are different from those by which the disease is usually recognized. They are certainly never those characterizing bilious remittent or intermittent fever. Relapses of the kind do not show themselves during the winter succeeding the season of the original attack, and we may look in vain for any fact justifying the belief that an attack of yellow fever in the autumn will render the system liable to intermittent fever the next spring. If now we turn to bilious remittent fever, we shall find that relapses, well characterized or obscure, are very common in it; that

¹ Dickson, pp. 344-5; A. Smith, op. cit., p. 60.

the disease, in these relapses, preserves the characters of the original attack—varying, perhaps, in type, not in nature—and certainly never assuming the garb of yellow fever; that these relapses continue sometimes to occur during winter, and are “multiplied sometimes to an epidemic degree in spring, under the name of vernal intermittent.”¹

Nor can we coincide with those who affirm that, in their symptomatology, the yellow and bilious remitting fevers approximate closely enough to be viewed as identical in nature, and differing only in respect to the degree of their violence or malignancy. As “in every febrile complaint there is an assemblage of phenomena which enables the experienced observer to ascertain its nature, and to discern its relations to other disorders,”² so the yellow fever is characterized by phenomena which distinguish it from the bilious, and prevent us from confounding them together. That, in the commencement of an attack, as I have already stated, the diagnosis cannot always be made out with precision, no one will be disposed to deny; for the opening stage of all febrile diseases, even the most dissimilar, is often marked by symptoms so near alike as to enhance the difficulty of discovering at once the true nature and character of the one of which they are the precursors. It is well known, too, that there are phenomena common to all fevers that arise from a zymotic source; and that in attacks of each of these fevers one or more phenomena, which, strictly speaking, do not appertain to it, but are characteristic of others, will occasionally intermingle from the effects of accidental complications—all of which will impede the facility of discrimination. Yet, in most instances, there is a sufficiency of means, even at the commencement of an attack, to enable the experienced and observant physician to form a correct opinion relative to the nature of the disease; while, as the latter advances, symptoms present themselves which render an error of diagnosis no longer possible. Even in that form of remittent fever which prevailed among the blacks of Philadelphia and New York in 1820, and which, from its appearing in the former city just prior to the breaking out of an epidemic of yellow fever, and coexisting for a while with the latter, besides bearing some resemblance to that disease, was at first thought to be identical with it in nature, the line of demarcation could easily be drawn. Thus, the late Dr. Joseph Klapp, in describing, under the name of typhus of the blacks, the cases received into the Philadelphia Almshouse, called attention to several points of difference between the two diseases, which, when viewed in connection with the facts that the fever in question was marked throughout its course by distinct daily remissions and exacerbations, presented no metaptosis or sudden cessation of febrile symptoms, commenced much earlier in the season than has ever been the case among us with regard to the yellow fever, prevailed among a race little obnoxious to the latter, and originated in, and was restricted to, localities which the yellow fever seldom visits, while it spared the ordinary haunts of the latter, leave no doubt on the subject. “1. In the typhus of the blacks,” he remarks, “in the very first stage, the epigastric region evidences great sensibility to the touch, whereas

¹ Drake, ii. 354-5.

² Smith on Epid., p. 109.

in the malignant yellow fever this sensibility does not usually occur until about the third day. The particular seat of this morbid sensibility is different in the two diseases; in the former it is more external, while in the latter it is chiefly internal. 2. In the typhus, the most active, as well as the most unpleasant remedies are generally retained with ease on the stomach; not so in the yellow fever. 3. In the different stages of the typhus, the tongue undergoes the several changes characteristic of that disease, but in the yellow fever it presents a different appearance; in no case of the latter disease have I met with the hard, dry, and glossy tongue, the usual attendant on the former. 4. Hemorrhages, in the second and last stages of the typhus, are uncommon, but very common in yellow fever. 5. In the typhus, there is an early prostration of muscular power; not so in yellow fever. 6. The yellow or icteric eye marks the first stage of the typhus, but in yellow fever it rarely occurs before the third day. 7. Black vomit is very common in the second and last stages of yellow fever, and very uncommon in the typhus of the coloured people. 8. The convalescent state of yellow fever is generally short, and as generally tedious in the typhus.”¹

1. If the yellow and bilious remittent fevers were identically the same—if the former was only a higher grade and more aggravated form—in other words, the maximum—of the latter, we might expect to find that mild cases of the yellow fever were still worse, and consequently more fatal, than the worst form of the bilious remittent; experience, however, teaches us that such is far from being the case. There is no physician at all conversant with the two diseases who has not seen instances of the last mentioned fever hurrying the patient to the grave without manifesting the peculiar characteristic phenomena of the yellow fever; and, on the other hand, instances of the latter are of daily occurrence in almost all epidemics in which the disease is of so mild a character as scarcely to compel the patient to keep his bed, and to yield with facility to the operation of trifling medicines, though nevertheless presenting those peculiar features which serve to distinguish the true yellow fever. If the opinion in question were correct, such things could not occur. Mild cases of yellow fever would be more violent than the worst forms of the bilious remittent; and yet the latter—the reputed milder form of the two—is found to occasion death, while the former, or maximum form, soon terminates in recovery. This fact has been amply illustrated in all our epidemics, and in none more so than in 1820, when the prevalence of the black fever, already mentioned, and of the common country fevers, afforded additional opportunities of contrasting the two diseases in that respect. Dr. A. Smith, of Galveston, has, among others, particularly called attention to the same fact.

2. In epidemics of yellow fever, the first cases are almost always the most malignant and fatal. This occurs in cholera, in plague, and some other complaints. No such peculiarity attends the outbreak of fevers produced by ordinary paludal exhalations, whether common intermittents or remittents.

3. The yellow fever appears sometimes sporadically in places subjected to

¹ Med. Recorder, iv. 85.

epidemic manifestations of the disease. In very many instances, no such sporadic cases occur during a long succession of years; while intervals of longer or shorter duration occur between epidemics. Dr. A. Smith has well remarked, in relation to this matter, that "when (common) miasmatic diseases exist, they are endemic, and occur more or less every year, no year being exempt from cases." (*Op. cit.*, p. 55.)

4. The yellow and common autumnal fevers, though kindred zymotic diseases, are not, strictly speaking, convertible; though, as we have seen, cases occur which exhibit symptoms characteristic of both, and the diseases may blend together, or appear in rapid succession in the same subject. Remittents and intermittents never change into yellow fever by the natural process of conversion, and *vice versâ*. Dr. Ashbel Smith, in his excellent parallel of the two diseases, has stated the case very accurately. "From the mildest intermittent, amounting only to a sensation of *malaise*, recurring at regular intervals, to the most ferocious congestive fever, we trace a regular, uninterrupted gradation; and in my section of country it is common to see, in the same individual, one grade converted into or succeeded by another within the period of a single interval—that is, a mild intermittent become congestive, or assume the form of a malignant remittent—and, *vice versâ*, the two latter forms disappear in the shape of a common ague and fever, or a mild remittent. So in yellow fever, there is a regular gradation from the exceedingly mild febricula through all stages of severity to the most ferocious black vomit. We also see sudden conversions of grades in yellow fever similar to those mentioned as occurring in miasmatic fevers." "To sum up, instead of malarial fever running into yellow fever, the same parallelism from great mildness to extreme severity, with sudden conversions, not into each other, but into different grades of the same disease respectively, subsists in the two diseases" (p. 54). All instances of apparent conversion from yellow fever to the other form of disease in question, and the reverse, as well as cases of either in which the symptoms are associated with some of the phenomena of the other, are more properly to be attributed to the co-operation of the cause of both diseases, and the coexistence of these in the same subject. In other words, they are the results of the complication or blending of two separate and independent complaints, and must not be viewed, as is sometimes erroneously done, as manifestations of different forms of one and the same complaint.

A case commences with symptoms of common remittent or intermittent fever, and at its close exhibits phenomena appertaining to yellow fever. In other instances the reverse occurs, cases of yellow fever ending with symptoms of periodic fever. The same occurs in other diseases. Typhus, typhoid, or pestilential fevers terminate sometimes in the same way; while, at other times, cases which at their outset presented the characteristics of common intermittent or remittent fever, assume, as the disease progresses, those of the fevers mentioned. So also with regard to pneumonia and fever. Cases of the former not unfrequently, under peculiar endemic or epidemic influences, end with symptoms of autumnal, as also of yellow, or typhus, or typhoid

fever. On the other hand, cases of periodic or other fevers sometimes terminate with symptoms of pneumonia.

Doubtless changes of the kind may, strictly speaking, be regarded as the effect of conversion; but the conversion herein noticed is not that of one form of a disease into another form of the same. It cannot be occasioned by an increased force in the cause of the disease first existing, by a difference in the state of predisposition of the person attacked, or by the peculiar mode of treatment pursued. It is not the result of a mere modification of one and the same thing; but a change of one thing to another of a more or less different kind; in a word, it is the substitution, partial or complete, of one disease for another. Such conversions of diseases are not of rare occurrence. They are, indeed, familiar to all practitioners. They often lead to evil or fatal consequences, or simply to the removal of disease without restoration to health; and, without feeling disposed to believe, with Sir George Gibbes, that one disease is always necessary to the cure of another, that just as many functions undergo a secondary derangement as are necessary for the cure of the primary one, and that no diseases occur but such as are curative in their effects or in their tendency,¹ we cannot doubt, taught as we are by daily experience, that the cure of one disease is often effected by the occurrence of another; but whether leading to baneful or salutary consequences, while many conversions are due to the transfer from one part to another of a specific cause, floating, as it were, in the system, as the gouty, rheumatic, &c., a large number are the effects, not of the operation of such a cause acting in this latter way, but, as already stated, of the slow or abrupt substitution of one disease for another; sometimes, though not necessarily, allied to it in a pathological or etiological point of view.

5. The bilious remittent fever, as its very name imports, is remittent in character, and presents periods of decrease and exacerbation in the febrile excitement, sometimes once, at another twice in the twenty-four hours, and at others again every other day—each exacerbation being generally attended with a sensation of chilliness, or a regular chill. Unless arrested or modified by remedial means, or by an effort of nature, it runs its course in a progressive manner either to a happy or fatal termination, to the end of its allotted time, and presents none of the symptoms characteristic of the yellow fever. Exceptions to this are rare; and even in what is denominated a continued fever, the remissions, though slight, can generally be detected, even in the commencement, while they become distinct as the disease advances. In pure yellow fever, on the contrary, the one paroxysm of which the fever consists, is not invariably preceded by a chill, and is almost always continuous in type, or marked by very slight, obscure, and irregular abatements of febrile excitement rather than remissions, properly so called. If these are of a more distinct character, it is only in very mild cases; and in them, as in the others, they are associated with other symptoms which characterize the disease. Besides, instances marked by such symptoms, and presenting a

¹ Brit. and For. Med. Rev., xxiii. 591.

remittent type, are, in all probability, the results of complications. Were it not so, the existence, in yellow fever, of remissions, and even intermissions, would not prove the identity of the disease with the other forms of fever, remittance and intermittence, being pathological conditions not unfrequently attendant on diseases of dissimilar character.

6. The attack, in common autumnal fever, is in general gradual, and preceded, for some time, by premonitory symptoms. The first paroxysm is marked with a tendency to increase in violence. Nocturnal attacks are comparatively infrequent. In very many cases of yellow fever, on the contrary, the disease attacks suddenly, without premonitory ailment of any kind. It usually seizes on the patient during the night, and is often violent on the first day, the symptoms following each other in rapid succession, while the disease runs a shorter course.

7. The two diseases, though somewhat allied to each other in the circumstance that in both the period of incubation is, in ordinary cases, limited within much the same range, differ materially in regard to the time to which that process may extend. We have seen that in the yellow fever the ordinary length of the incubation is from five to nine days—that occasionally it has been prolonged to a month or two, and that in one solitary case only has it been known to extend much beyond that time. In autumnal fevers, the period of latency is seldom shorter than three or four days. In general it extends beyond this, and has not unfrequently been known to reach a limit far exceeding that noticed in the yellow fever. Dr. John Hunter informs us that the Suffolk militia were called, in 1793, from their healthy country to Hilson Barracks, the low, marshy, unhealthy situation of which is proverbial. Twenty-two died before they left at the end of June. In July, the regiment, with eleven other battalions, encamped at Watertown, near Tunbridge Wells. One hundred sickened soon, out of five hundred, with fever. Some were taken ill in October, or four months after leaving the Hilson Barracks. The 18th regiment, in 1783, after being at the same barracks from June 22 to October 9, were sent to Gibraltar. There were then sixteen of the men labouring under ague. While at Gibraltar, though the regiment was only four hundred strong, the disease spread so rapidly among them that, by May, the cases amounted to two hundred and eighty (including women and children), of whom a part were then recently attacked for the first time. Whilst such was the course of events in this ill-fated regiment, the disease did not exist in any other part of the garrison.¹ Dr. Hunter adds that “ships, returning from a warm climate, particularly if they have been in harbour during the unhealthy season, have many of their men taken ill of the remittent fever, even two or three months after being at sea.

Some of the British soldiers who inhaled the pestiferous atmosphere of the Walcheren Marshes, were attacked for the first time in healthy situations in England—Colchester, Woodbridge, &c.—as late as nine months after they

¹ *Op. cit.*, p. 335.

were brought back.¹ The following facts, communicated to Dr. Bancroft by Mr. Nixon, surgeon to a battalion of the 1st Regiment of Foot Guards, will be read with interest. The battalion landed on South Beveland on the 2d of August, 872 strong. On the 19th, the endemic appeared among the men, and between that day and the 4th of September, when the men embarked for England, *i. e.* sixteen days, 359 of them were attacked. The battalion was landed at Chatham about the 7th or 8th of September. Many of the men continued to be attacked with endemic fever, so that, by the 8th of March, 1810, only 117 of the original strength had escaped the disease in question. Some of the 117 men were attacked with intermittent fever as late as the middle of the month of June.² Dr. Férrus, a distinguished physician of Paris, relates a striking instance of the kind. Three hundred men, of the old Imperial Guard, to which he was surgeon at the time, were exposed to the cause of autumnal fevers in Breslau. Many of them took the disease ten days after leaving the place; other cases followed, and the fever became general. Dr. Férrus himself was attacked six months after, while stationed on the Niemen, where no disease of the kind prevailed, and at a period when the country all around was perfectly healthy.³ Of forty cases of intermittent fever, which occurred on board the H. C. ship *Barrosa*, in 1832, 1833, three took place seventy to eighty days after leaving England; thirty-one while lying at Whampoa; and seven from two to three months after leaving Canton, and ninety-three days after the disease had ceased in the ship.⁴ Labourers, especially the Irish, will go down, for harvest work, into Lincolnshire, and bring back the seeds of ague within them, and yet may not be attacked for weeks or months.⁵ M. Boudin calls attention to a fact which he himself noticed; that regiments that had returned to Marseilles, where periodic fevers are but little known, from the malarial districts of Algeria and Corsica, as well as those that had formed part of the expeditionary army of the Morea, continued during several years (*des années entières*) to suffer from diseases bearing unequivocal marks of the fevers of the localities they had left.⁶ Towards the close of the year 1843, two regiments of infantry arrived at Courbevoie; the one (23d Light Infantry) coming from a northern garrison, the other from the citadel of Strasburg, where malarial fevers prevail extensively. The two regiments occupied the same barracks, performed the same duties, partook of the same fare, and were in every other respect on the same footing. Yet, while the first of these regiments suffered from typhoid fever and pneumonic inflammations, the other furnished, for more than a year, a large number of intermittent fever cases. The disease spread to several hundred of the men, and spared few—not even those who had not had it during their stay in the malarious locality whence they came.⁷ A similar

¹ Bancroft on Yellow Fever, pp. 241, 304; Blane, Diss., i. 244; Williams, Morbid Poisons, ii. 465.

² Bancroft, pp. 307, 308, note.

³ Diction. de Méd., xii. 6; Ibid., 2d ed., xviii. 69.

⁴ Peterson, Med. Gaz., xv. 269.

⁶ Annales d'Hygiène, xxxiii. 63.

⁵ Med. Gaz., xxviii. 365.

⁷ Ibid., p. 65.

occurrence was observed in another regiment (the 75th) transferred from Strasburg to Versailles, in the autumn of 1843. In December of the year following, the fever was still prevailing, but only among the men who had arrived from Strasburg; the new recruits remaining free from the disease.¹ In another essay, M. Boudin remarks: "So far as regards myself, after a survey of the numerous observations we have collected in France at periods and in localities exempt from periodic fevers among men arriving from the paludal districts of Corsica, Morea, and Africa, I have no hesitation in declaring that the period of latency of the malarial intoxication may extend to eighteen months."²

One of our countrymen, Dr. L. H. Anderson, of Alabama, in a Prize Essay, already referred to, states that he was himself attacked in the city of Paris, six months after leaving a mountainous district, with an intermittent of a very different character from the fever usually observed in that city. The remote cause, as he remarks, had no doubt been harboured in the system all the time, his general health appearing, notwithstanding, for three months before the attack, better than it had been for years.

8. The yellow fever is, as has been several times stated, a disease of one paroxysm, which continues one, two, or three days, and is followed by a total cessation of febrile symptoms—not a remission—and this again by prostration. This state of apyrexia, though usually marked by a moist natural skin, a diminution of heat, and an entire absence of all pain, is not always the precursor of recovery—as occurs in most other fevers—or followed by periodical exacerbations of febrile excitement. It is, in fact, an alarming state of collapse. This metaptosis occurs in almost all cases—those that end in death as well as those that end in recovery—and is not, therefore, characteristic of fatal cases alone. Nothing like this occurs in the bilious remittent form of fever. In this disease, the periods of abatement are followed by periodical returns of arterial excitement, and these alternate till the final termination of the case. If an intermission occurs, it is always to be viewed as the forerunner of convalescence, or the conversion of the disease into an intermittent fever; while if, after an intermission has occurred, symptoms of serious import present themselves, the change must be viewed as the effect of an accidental relapse.

9. The yellow fever runs its course, in general, more rapidly than the bilious remittent, ending most frequently in three, five, or seven days. The other seldom ends in less than from nine to twenty-one days. This cannot be owing to the former being simply a more violent grade of the latter, inasmuch as the milder cases of yellow fever, which, on that supposition, would still be more violent than severe cases of bilious fever, present the character of a mere ephamera, and are much shorter in duration than fatal, or even mild and trifling, cases of the other.

10. The discoloration of the skin presents some differences in the two diseases, as to the frequency of its occurrence, the character of its hue, and the

¹ Loc. cit., p. 64.

² Boudin, *Géographie Méd.*, p. 64.

rapidity of both its diffusion and disappearance. In the yellow fever, it is often, though not invariably, noticed; appearing, in most cases (except those that prove quickly fatal), either during life or after death—almost universally at the latter period. In the bilious remittent, jaundice is very often absent. In the yellow fever, it assumes sometimes a pale lemon, or deep orange, but most commonly a yellowish, dingy brown, or even mahogany or livid hue, and is often darker after death than it was during life; whereas, in bilious remittent fever, when it occurs, it is more complete, and differs in nothing from the hue of common jaundice—of a lighter or deeper shade. In the yellow fever, it disappears rapidly—often with remarkable rapidity; in the bilious remittent, the reverse is usually noticed. In the yellow fever, it diffuses itself somewhat quickly; in the other, its progress is slow. In the yellow fever, jaundice, when it occurs early, is always a sign of the gravest import—often the harbinger of death; in the other disease, the yellow discoloration of the skin is always innocuous.

11. The peculiar swollen, suffused, inflamed, turbid, watery, and glassy appearance of the adnata observed in the early stage of the yellow fever—so easily recognized, and so difficult to describe; and, when once seen, never to be forgotten—and its peculiar yellow tinge in the after period of the disease, are seldom seen in the bilious remittent. When this tinge is perceived, it is not as well marked as it is in the former. In the bilious fever, the eye, in the first stage, presents a more fiery, but less glassy and ecchymosed redness; while the icteric colour of that organ, which, in the other, never, or very seldom, appears before the cessation of the fever, comes on sometimes in the early stage, and assumes in the latter a deeper saffron hue.

12. Among the symptoms which characterize the yellow fever, even in its mildest forms, I have already mentioned a peculiarly severe and constant pain situated in the supra-orbital region, or at the bottom of the orbits, and, apparently, unconnected with disordered action of the brain. In the bilious fever, the headache is seldom of this kind, even in the more serious attacks; it is more generally diffused over the forehead or top, and sometimes occupies the back part of the head, and is attended with a sense of fulness and other marks of disordered cerebral circulation. The yellow fever may also be distinguished by the severe lumbar pain noted in most, if not all, cases; and also by the pain in the knees and calves of the legs, cramps, &c., all of which are considerably more severely felt in this than in the other form of fever; in which, indeed, they often fail to appear, and are almost always quite moderate.

13. From the state of the intellectual faculties in these two diseases, we may derive an argument against the opinion of their identity. In the yellow fever, as we have seen, the mind is often unimpaired from first to last; or, after having been affected during the stage of excitement, it ceases to be so, and continues free from disorder to near the fatal issue—often to the very last; whereas, in a large number of instances of bilious remittent fever, the intellect, though not always affected in the early stage, soon becomes more or less involved in the disorder of the system, and, when once affected, continues so, to a greater or less extent, to the end, unless relieved through the effect

of treatment. It varies often with the different phases of the disease—lessening with the morning remission, and increasing with the exacerbation. The great flow of spirits which sometimes marks the first stage of the yellow fever—the anxious fear on the one hand, or the perfect indifference or callousness on the other, evinced by many, appear peculiar to the yellow fever; or are not often noticed, to any very great extent, in the other form of fever. When delirium does occur in the yellow fever, it is generally of a somewhat different kind from that experienced in the bilious remittent. In the latter, though sometimes violent, it is more generally mild, showing itself rather by a great confusion of thought than by violence of action or great incoherency of language. In this disease, we often notice, as stated, a rapid and unconnected succession of ideas, over which the patient can exert no control. In the delirium of yellow fever, we often find the patient entertaining some fixed ideas deeply impressed on the mind, which he has the power of retaining before him, or which, apart from consciousness or the will, remain fixed in the mind; and, reasoning from which, he arrives at correct conclusions.

14. Another peculiarity of the yellow fever is the unimpairedness, in very many cases, of the muscular strength, even to the last moment. Nothing of the sort is noticed in the bilious remittent fever, in which muscular debility is one of the earliest, most constant, and marked symptoms, prevailing from the onset of the attack, and progressing up to the termination of the case.

15. One of the most usual and common symptoms of the bilious remittent fever is a thickly furred yellow or brownish tongue, the edges being at the same time more or less red, and indented by the teeth in consequence of being somewhat swollen. The organ is occasionally rather moist, and continues in this state for some time, becoming dryer and darker as the danger increases. In many cases it is blackish, especially in the centre, and rough or fissured on the surface. It assumes a cleaner and paler appearance as convalescence approaches, the change commencing at the edges and extending towards the centre. In the yellow fever, it is not uncommon to find the tongue clean and of perfectly natural appearance. In other cases, it is smeared over, as it were, with a thin coating of white or yellowish mucus; while the edges and tip are very red, and the latter is pointed and trembling. It is often, at the same time, moist. In this state the organ continues often to the last, whatever may be the issue. In other cases, as the disease advances, considerable changes take place in the appearance of the tongue, which, having been fully described in a former chapter, need not be mentioned here. They mostly contrast with those observed in bilious remittent fever. Thirst in bilious remittent fever is very frequently, though not constantly, noticed; often it is moderate, except during the exacerbation, and it generally harmonizes with the condition of the tongue. In the yellow fever, thirst, though sometimes moderate, or even almost absent, is generally considerable, and even intense; while the harmony above alluded to does not exist, a moist and natural tongue being associated with an unquenchable craving for drink, or the reverse.

16. The pulse and temperature of the skin are sometimes little or not at all altered in the yellow fever; or, while the circulation is highly excited, as

marked by a quick pulse, the skin remains cool and moist, and is often preternaturally cold. Such phenomena are not characteristic of bilious remittent fever, in which, except in cases attended with congestion, the pulse is always more or less accelerated, full, strong, or contracted, and the skin hot and dry. When, in the latter stage, the surface becomes cool or cold, it is at the same time, particularly about the extremities, bedewed with sweat, more or less copious in quantity, and of a clammy character. In the last stages of the yellow fever, especially in fatal cases, the pulse lessens in velocity, and often becomes very slow, unless quickened by stimuli, and often in spite of these. In the bilious remittent fever, the lessened quickness of the pulse is usually a favourable sign; while in severe and fatal cases the pulse, instead of becoming slow, increases in rapidity as the disease advances. As Dr. A. Smith remarks, in the latter disease it is rare to find the pulse below the healthy standard of frequency, except after the complete abatement of the disease.

17. The two diseases differ widely as regards the nature of the matter vomited, the manner in which it is ejected, the phenomena by which the emesis is accompanied, and the effect of remedies upon it. In the yellow fever, nausea and vomiting are, in almost all cases, early and distressing symptoms; and, instead of being benefited or removed by emetics, are aggravated by them. At the onset, the matter thrown up consists, besides the contents of the stomach, of glairy mucus, mixed, in some cases, though not in all, with more or less bile. As the disease advances, bilious discharges cease, and are replaced, in most instances, though not in all, by the matter so well known under the name of black vomit, which, as we have seen, is not bile. In the last stage of this disease, the ejection takes place by jets, as if by a sudden spasmodic contraction of the stomach, of which the patient is not forewarned, and over which he has no control; while he suffers from a burning heat at the præcordia and along the œsophagus, and from the acid and acrid taste of the matter ejected. In the bilious remittent, the stomach may indeed be affected, but it is so, generally at least, in a milder degree. The discharges usually consist of bilious matter, mixed at times with the contents of the stomach and mucus; and though, in some instances, black matter, similar to that observed in the yellow fever, is thrown up, the occurrence is very rare; so rare, indeed, that physicians of large experience may be, and have been, years without seeing a solitary instance of the kind. Being, besides, observed sometimes in diseases of very dissimilar nature, the occurrence cannot be adduced as a proof of the identity of the two fevers, but only as an adventitious circumstance, of no value in a diagnostic point of view. Nor is it less true that, in many cases, the black discharges said to resemble those of yellow fever will, if properly examined, be found to consist of bile and dark but unaltered blood; and, again, that cases in which real coffee-ground matter was ejected, and which were represented as of a bilious remittent character, were, in truth, instances of true yellow fever. In remittent fever, the mouth is bitter, and the matter ejected of the same taste; while the act of vomiting is preceded by nausea, and is never the result of the sudden con-

traction of the stomach above adverted to. There is less, if any, burning sensation along the œsophagus, fauces, or at the epigastrium, and the affection of the stomach, in the early stage, is usually, or frequently, removed by clearing out the organ by means of emetics.

18. In yellow fever, tenderness at the epigastric region is seldom experienced before the third day; whereas, it is often noticed in the early stage of the bilious remittent. In the black or negro fever of 1820 and 1821, as already stated, it was particularly observed to occur very early, and to be much more acute than in the yellow fever.

19. It has already been stated that, in some cases of yellow fever, the mind and sensibility being unimpaired, the appetite and relish for food return some moments before death. The patient sits up in bed with ease, or even gets up, and eats with much delight, though the pulse at the wrist has disappeared, the coldness of the surface portends the approach of death, and the countenance is hippocratic. No such results obtain in fatal cases of bilious remittent fever, in which a return of appetite is always attended with a favourable change in the other symptoms, and indicates the approach of convalescence.

20. In bilious remittent fever, the secretion of bile is usually increased, and continues to the end, as evidenced by the nature and quantity of the matter ejected from the stomach, or passed off by stool. In the yellow fever, on the contrary, the secretion of bile is more or less diminished from the onset of the attack, and becomes entirely suppressed in the advanced period of the disease. In the one case, a diminution in the quantity secreted is considered a sign of good omen, whereas in the other the reappearance of bile in the stools is regarded as a favourable symptom. "If I were asked what was the most prominent pathognomonic symptom of yellow fever, one that most distinguishes it from those cases of remittent bilious fever complicated with gastritis so prevalent during epidemics of the former, I would answer, the total cessation, or the much diminished and vitiated secretion of bile. For although it is sometimes ushered in by bilious vomiting, the bile thus ejected has been mechanically forced from the gall-bladder where it had been collected previous to the attack. It is very certain that the restoration of the functions of the liver is the most favourable symptom in the course of the disease."¹ But even were bilious symptoms more common than we find them to be in yellow fever, the circumstance would no more prove this disease to be nothing but a high grade of the bilious fever, than their occurring in irritations of the stomach, from common or external causes, would prove these irritations to be identical with this form of disease. Bilious phenomena, in fact, may be associated with symptoms of complaints of a dissimilar character, and can very readily be understood to be capable of combining with those of the yellow fever.

21. The condition of the urine is not exactly the same in the two diseases. In the bilious remittent, the fluid is commonly of a reddish, or more or less

¹ Wurdeman, Am. Journ., N. S., ix. 51.

deep-yellow hue, and scanty. Sometimes it is copious and limpid. As the disease advances, it generally becomes turbid, and at the close, especially at the period of crisis, becomes sedimentous and deposits a quantity of lateritious matter. As is known, this fluid in its normal state contains, on an average, 11.88 of urea, 0.395 of uric acid, 6.80 of inorganic salts, and 8.60 of organic matter. Of the salts, the chloride of sodium may be estimated at from $3\frac{600}{1000}$ parts to $4\frac{45}{100}$ in 1,000. Now when, with this before us, we inquire how matters stand in respect to remittent and other malarial fevers, we find that these diseases differ in no inconsiderable degree from the state of health, so far as the proportionate quantity of those ingredients is concerned. In them the urea is in less quantity than in health, presenting a proportion of only 9.01. At the same time, the quantity of uric acid attains an amount of 9.80, or eight times larger than in health. As regards the inorganic salts, we find them to rise as high as about 16.72, or much more than twice as high as in healthy urine, while the organic matter varies but slightly, if at all, from the normal state. As a matter of course, amid this increase in the salts, the chloride of sodium can suffer no diminution, and, in all probability, has increased in the same ratio as the other salts.¹ During the stage of apyrexia in intermittent fever, the urine is often found, so far as the above ingredients are concerned, in its natural state; a condition proportioned in great measure to the extended duration of that stage and the shortness of the febrile paroxysm. In some cases, the fluid is healthy throughout. But, in most instances, the changes under consideration exist as well in the intermissions as during the paroxysms, and are greater in proportion to the length of the disease. In no instance can we discover that the proportion of uric acid remains at the normal point, and that the quantity of the fixed salts falls considerably below.

In the yellow fever, the urine is often unchanged in the early part of the attack, though as often high coloured and scanty. As the disease advances, it assumes a saffron colour, depending on the presence of the colouring matter of the bile, and is found very deficient in the due quantity of urea, and to contain little or no uric acid. It is almost always albuminous.

The bladder, in the latter disease, is often insensible to distension—which is not the case in the other.

22. Suppression of urine from a want of renal secretion is, as already seen, a common attendant on the last or fatal stage of the yellow fever; and marks, in an almost unerring manner, the approach of death. In the bilious remittent, this symptom is seldom observed, or not more frequently than in the phlegmasiæ.

23. Petechiæ and vibices are often, and carbuncles and buboes are sometimes observed in the yellow fever, and serve, to a certain extent, to distinguish that disease from the bilious remittent, in which the first two are seldom, and the others never noticed.

¹ Becquerel, *Séméiotique des Urines*, pp. 286, 291; Heretier, *Chimie Méd.*, p. 528; Simon, *op. cit.*, ii. 255, 257.

24. The pearl-like coating of the gums which, as we have seen, is very often, though not, perhaps, invariably observed in the yellow fever, has not, so far as I am aware, been found in bilious remittent or intermittent fever.

25. The state of the venous blood, drawn during life, and of that found after death in the heart and large vessels, differs somewhat in the two diseases. It is very florid in the early stage of some cases of the yellow fever; but in general it is dark in colour, homogeneous in appearance, and possesses little if any power of coagulability. As the disease advances, the fluid becomes darker and less coagulable; while the serum assumes a yellow colour from the presence of uneliminated bilious colouring matter. It is seldom sily, and if so, covered with a soft pellicle. In a very few cases, it is buffy. In the bilious remittent, though oftener sily and sometimes cupped in the early stage, from the more frequent occurrence of local inflammation, the blood has seldom the appearance of arterial blood; and, at a subsequent period, presents but very rarely to the same extent the appearances of dissolution noticed in the yellow fever, unless complicated with a typhous condition of the system, and seldom assumes the dark yellow tinge mentioned above.

26. The hemorrhagic tendency is different in the two diseases. In the yellow fever it is well marked, and constitutes, indeed, one of its main characteristics; for, besides the black vomit, we find the blood oozing from all the outlets of the body. In some cases, the blood oozes from the skin, eyes, and ears. It is extravasated into the cellular membrane, or even into the substance of the muscles, where it collects in large amount. All this is extremely rare—much of it, indeed, not seen in bilious remittent fever. Hemorrhage from the gums rarely occurs unless it be in cases in which mercurial salivation has been brought on, and it may be safe to say that effusion of blood from the eyes, ears, skin, or into the cellular tissue and the muscles never occurs.

27. The livid patches, and other morbid states of the skin noted in the yellow fever, are never or seldom seen in the bilious remittent fever. The same may be said of the affection of the testes—the diminished size of these—their retraction towards the abdomen—the flaccid feel, emptiness, and excoriation of the scrotum—the discharge, in some fatal cases, of a bloody, ichorous, and fetid matter from the urethra.

28. The convalescence in yellow fever is usually, under proper management, rapid; the disease rarely entails sequelæ of much importance, whilst relapses, properly speaking, are not frequently encountered. The reverse, in all these respects, is not uncommonly the case in the bilious remittent fever, in which convalescence is, in many cases, protracted, from which the patient rises with enlarged spleen and diseased liver, with the concomitants of these—dyspepsia, dropsy, jaundice, and intestinal disorder—and in which relapses are very easily excited.

29. The venereal appetite, spontaneous and genuine, is aroused in the convalescence of some cases of yellow fever. It sometimes manifests itself in the

stage of extreme prostration. I have seen or heard of nothing of the kind in bilious remittent fever.

30. The idea of specifieness does not necessarily carry along with it that of the existence of contagion, for there are more morbid poisons than one, producing local and general effects of a peculiar and specific character, which do not reproduce their kind by direct or indirect contact. The two diseases being non-contagious is, therefore, no proof of their depending on the same pathological state, and of their being the result of the same cause.

31. Dissections reveal somewhat different phenomena in yellow and bilious remittent fevers. In both, the stomach sometimes exhibits marks of inflammation; but, in the latter, the flea-bite and mammillated appearances seldom if ever occur, and the organ but very rarely contains the matter of true black vomit or pure blood; while, after yellow fever, it exhibits those morbid changes in many cases, and almost invariably contains more or less of the matter above mentioned. The intestines, in remittent fever, are likewise congested; but, very seldom contain a black or tar-like matter similar to that found in the yellow fever. The matter, when dark, consists of altered bile. The Peyerian glands, if we are to credit some writers, are not unfrequently affected in the bilious remittent. As a general rule, they are never so in the other diseases. The liver in the yellow fever is generally, if not always, of a yellowish colour, dry and anemic in texture, and filled with oily matter. In the bilious remittent it is often enlarged, generally soft, more or less engorged, and of a bronze or slate colour externally, and of a uniform olive or light bronze upon its cut surfaces.¹ My very distinguished friend, Professor Alonzo Clark, of New York, to whom, as stated in a former chapter, credit is due for first calling attention to the fatty condition of the liver in the yellow fever, has obligingly favoured me with the results of his researches into the state of the same organ in the bilious remittent. In this interesting and valuable communication—which I cannot resist the temptation of inserting entire, and which will be found at the end of the present volume—Dr. Clark remarks that, to the naked eye, the peculiar hue alluded to seems to be nearly uniform, as if produced by a dyeing process, which had stained alike each particular fibre and particle of the organ. But when a thin section of such a liver is examined microscopically, it is easy to see that its surface is sprinkled over, and its whole thickness dotted everywhere, with dark-coloured particles of irregular size and shape, totally different from anything that enters into the healthy constitution of this organ. “A little closer examination demonstrates that this adventitious matter occupies many of the secreting cells, and even sometimes the nuclei of these cells, while it is, at the same time, diffused throughout every part of the tissue, apparently without rule or order—unless, perhaps, it be noted that it is most abundant near certain bloodvessels.” This adventitious deposit—the result of some modification of hæmatin—is not found in the

¹ Stewardson, *Am. J., N. S.*, i. 289; Swett, *ib.*, ix. 29; Anderson and Frick, *ib.*, xi. 312; A. Smith, *Tr. of N. Y. Acad. of Med.*, i. pp. 65–6.

liver of the yellow fever—at least, such has been the result in the examinations made by Dr. Leidy and others in this city, and by Dr. Clark, in New York. It is found abundantly in the black vomit, and not in the matter ejected from the stomach, or voided by stool in remittent fever; and while in the yellow fever the liver is filled with oil-globules, the deposit of them in the cells and tissues of that organ during remittent fever, though sometimes occurring, is not as uniformly seen as it is in the other disease—failing to appear in a very large number of cases, and when appearing being much less abundant—generally in no greater quantity than is found in the healthy or ordinary condition of the organ.

The gall bladder, in the latter disease, is usually distended with black bile. In the yellow fever it is seldom distended, and often contains less bile than in health. The spleen, in this disease, is often little if at all changed. In bilious remittent fever it is very generally much enlarged and softened. In the former, the kidneys are occasionally of a yellow colour externally, their substance congested with minute abscesses in the papillæ. I am not aware that such a morbid condition, or any of those noticed in a former chapter, has been found after the bilious remittent fever. In the latter, hemorrhagic infiltrations in the cellular membrane, in the substance of the muscles and in various organs are rare, and blackish spots or masses in the lungs—dependent, in part, upon the effusion of blood into their tissue—are seldom if ever seen. In the yellow fever, as already stated, they are common.

NOTE ON THE “BRONZE LIVER” OF REMITTENT FEVER.

MY DEAR DOCTOR: I do not know that I have a memorandum of any kind, relating to the pathological anatomy of remittent fever. For several years, as often as I have had an opportunity for the examination, my attention has been particularly fixed on the cause of the bronze, slate, or olive colour of the liver in that disease. It is to this point, I suppose, that your inquiries refer; and if there is anything in my investigations worthy of your notice, I cheerfully submit it to your charitable consideration.

The microscope, it may be safely said, carries us one step forward in our attempt to find the cause of this singular colour. To the naked eye, the peculiar hue seems to be nearly uniform, as if produced by a dyeing process, which had stained alike each particular fibre and particle of the organ. But, when a thin section of such a liver is examined microscopically, it is easy to see that its surface is sprinkled over, and its whole thickness dotted everywhere with dark-coloured particles of irregular size and shape, totally different from anything that enters into the healthy constitution of this organ. A little closer examination demonstrates that this adventitious matter occupies many of the secreting cells, and even sometimes the nuclei of these cells; while it is, at the same time, diffused

throughout every part of the tissue, apparently without rule or order, unless, perhaps, it be noted that it is most abundant near certain bloodvessels.

This adventitious deposit is no new thing to those who use the microscope. It is abundant in the black vomit of yellow fever. It is often found in kidneys which are the seat of the degeneration that produces albuminuria, in rapidly growing cancers, and, in general, in parts which have long been hyperæmic, even though not actually inflamed. In chronic inflammation of the large intestines and of the bladder it is deposited in patches, producing the dark spots so familiar to pathologists. It is often seen in bloody urine, and also in the urine of those suffering from Bright's kidney; in apoplexies, in the dark-coloured spots so often found in the ovaries. It is the *pseudo-melanose* or *éléments hématiques* of Lebert; when crystalline, the *hematoïdin* of Virchow. I have spoken of it in "A Note on the Microscopical Constituents of Black Vomit," &c. (*N. Y. Med. Times*, May, 1853), as the melanine or melanose of some authors.

The colour of this deposit is not uniform. A portion of it is as black and as opaque as charcoal. Some of it is of a semitransparent dark brown; and it is found of all shades, from red or orange to an opaque jet black. The size of the particles also varies. As seen with a magnifying power of 400 diameters, much of it appears as points or dots; some particles seem to be as large as the head of a pin, and the black matter is occasionally seen having the apparent size of the finger-nail and the form of an irregular flat scale. The shape of the smaller particles is commonly globular, if the colour be red or dark brown; but usually irregular if the colour be jet black, as if it had been broken from a larger thin scale. The shape of the larger black scales is infinitely irregular, and yet their form and the sharpness of their angles has suggested the term *crystalloid* as applicable to them (*Lebert*), or rather to the same material common in the lungs. If these scales have any well-defined border, they will on that border show a line of refracted light, just outside the opaque mass, which may appear like an investing cyst. These dense black scales are subject to fracture, but not at fixed angles.

Two varieties of colouring matter are possibly embraced in the description here given; or possibly a change is begun in the red and brown particles, which is completed in the black. This at least appears evident, that the red and dark brown semitransparent globular particles are slowly soluble in nitric acid, while the jet black friable substance is not in any manner affected by it. Yet it must not be forgotten that the red, dark brown, and black deposits are almost constantly associated, at least in parts affected with hyperæmia. I can hardly doubt that they are all the result of some chemical change or modification of hæmatin. Regarding the red and brown matter, this is conceded by all authorities, but whether the black particles and scales can have a similar origin does not appear to have been a subject of inquiry. This black matter appears to be identical with that so often found in the lungs of aged persons, commonly regarded as carbonaceous, and denominated *melanine* by Scherer, Robin, and others. The striking resemblance which it bears to a portion of the dust which falls in a room when anthracite coal is burned, may readily suggest the idea that it is some form of carbon; as has the opinion that it is most abundant in the lungs of those who work in coal. The analysis by Melsens, Schmidt, and Scherer, shows that it is not pure carbon. According to these authorities, it is composed of variable proportions of carbon, hydrogen, nitrogen, and oxygen (C, 58 to 96 per cent.; H, .1 to 7 per cent.; N, 3 to 13 per cent.; and O, from a small quantity, not stated, to 22 per cent.). It seems probable, from these analyses, that the pulmonary black matter is formed by the union of some highly carbonized matter with a protein substance. At least

the mechanical theory, which attempts to explain this deposit by the inhalation of solid particles of carbon, I can neither entertain nor understand. I could be much more easily persuaded that the carbonic acid, which so abounds in these organs, has some agency in producing it. Whatever theory satisfactorily explains the source of this material in the lungs, will doubtless apply equally to all the tissues in which it occurs; though in the first it hardly marks a disease, while in the liver it has rarely been found in any abundance, except after remittent fever.

I have said above that these two varieties of colouring matter are almost constantly associated. I will now add that in every organ, except perhaps the lungs, (these organs have not been examined with reference to this point), the orange-coloured particles pass by insensible shades into the reddish brown; and these, in the same way, into the dark brown; and the latter still, in the same manner, into the opaque black and friable substance. This will be distinctly seen on examining almost any part where hematoïdin, in any form, exists at all abundantly, by comparing different portions of it. But, what is probably still more to the point, scales are occasionally seen, having the crystalloid form and the friable character of the jet black matter, in which there is some translucency, and the deep reddish brown colour that marks the other variety. These have every property of the black matter, except colour and opacity. Scales are also seen which are red and granular on one edge, while on the other they are of an opaque black, and show the line of refracted light which is commonly seen only in the black scales. These facts go far to persuade me that the black matter is the completion of a change, or series of changes, which is begun in the lighter hues, and is seen advancing in the brown matter.

The *crystalline* red and brown matter is commonly associated with the other forms above described. Lebert, in his *Anatomie Pathologique*, now in course of publication, groups them thus: *les éléments hématiques, amorphes, granuleux, globuleux et cristallins, d'origine hémorrhagique*. In two remittent fever livers out of three, lately re-examined, the crystalline form occurs with the amorphous, granular, and globular. Its colour and association both indicate for it the same origin. It was to the crystalline variety that Virchow gave the name hematoïdin; but, persuaded of the substantial identity of all the forms, I have thought it safe to extend the application of the term so as to embrace them all.

How this colouring matter finds its way into the cells and substance of the liver during remittent fever, we can at present only conjecture; but, in forming an opinion on this point, the following facts are not unimportant:—

1. A deposit of oil-globules in the cells and tissue of the liver, during remittent fever, is not uncommon. It is not so frequently met with, nor is it so abundant, as in yellow fever; but I have just risen from the examination of three bronze livers of remittent fever (all that I have preserved), and I find that in two of them an unusual amount of oil is associated with the pigment matter. This fatty degeneration is often, I think, dependent on a low grade of inflammation, or at least a hyperæmic condition of the part in which it occurs.

2. The change which occurs in the liver, having for its symptomatic manifestation jaundice or a dingy hue of the skin in most cases, is not fully described by stating the colour of the organ. Besides the olive, slate, or bronze hue, it is often found enlarged, and, to a limited extent, softened; at the same time, in a few instances, marked engorgement of the vessels has been noticed; though, in most, if such engorgement existed during life, it was not remarked after death—it had disappeared, as it does in all mere hyperæmic conditions of the surface, whether inflammatory or congestive.

3. There are many conditions of disease in which the colouring matter of the

blood-corpuscles is yielded during life no less than after death, and stains the adjacent tissues. This occurrence is a part of the recognized history of inflammatory stasis. It is seen, to a remarkable extent, within the intestinal mucous membrane in cholera. It perhaps reaches its maximum in the same membrane from starvation. It occurs in the mesenteric glands when persons have been long nourished by nutritive enemata; and in many hyperæmic and inflammatory states. The fibrinous effusion of hemorrhagic pericarditis derives its red colour not from the blood proper, but by the imbibition of the hæmatin only. As a post-mortem appearance, the staining of the endocardium and large vessels by imbibition of hæmatin, in typhus and typhoid diseases, in scurvy, and, after a certain time has elapsed, in all diseases, is too well known to require description. This fact has been often remarked in the post-mortem examinations, early made, of those who have died of remittent fever.

4. In the black vomit of yellow fever, the blood effused into the stomach appears to be subjected to a chemical action, which not only converts the free hæmatin into *hematoïdin* or *éléments hématiques*, but produces a similar change in that which is retained by the corpuscles, giving to these latter a dark brown colour, and completely altering their chemical relations. (See *N. Y. Med. Times*, May, 1853.) The production of this colouring matter, to a limited extent, is a common occurrence in the stomach. Increased vascularity of the lining membrane is very apt to be followed by it, whatever may be the cause of the hyperæmia.

In view of these facts, we may conjecture that the morbid changes necessary to produce these colouring elements are partly hæmic and intravascular, and partly extravascular; that during remittent fever there is a hyperæmic condition of the vessels of the liver, falling short of inflammation, which does not leave constant traces after death; and that, at the same time, the blood in the vessels is in a condition to yield with facility its hæmatin; that this is extravasated, and, penetrating the cells and tissue, there meets with some fluid or gaseous matter which converts it into the various forms of *hematoïdin*. The occasional occurrence of the bronze hue in portions of the liver which have lain in contact with the intestines, in nearly every form of disease, is perhaps of some importance in this connection. This colour is ascribed almost universally to the influence of the intestinal gases. But the intestinal gases must find something in the liver to act on that does not exist in the intestinal coats, for these do not receive the colour which the gases are supposed to produce in the liver. This something is, probably, the free hæmatin from the circulating blood. The agent which produces the change in this hæmatin is as likely to be carbonic acid gas, in a nascent or mature condition, as any other known substance.

But conjectures, however plausible, are not *science*. They are mainly useful when they stimulate inquiry. The speculations here presented are offered in the hope that they will produce their legitimate fruit among those whose opportunities for investigating the lesions of remittent fever are more extensive than my own.

Regarding the importance of the bronze colour of the liver as the anatomical lesion of remittent fever, I have but little to say. The hospital with which I am connected, though a very large one, does not receive many cases of this disease; and within the paved and sewered districts of this island it is virtually extinct. In the last seven years, I may have witnessed seven post-mortem examinations; and though I have not kept notes of them, I can trust my recollection to assert that I have seen no instance in which one of the recognized hues did not exist. Portions of the liver, in each one of these cases, have been studied with the aid of the mi-

croscope, and in all, the colouring matter here called hematoïdin, has been found abundant; and in each case, as far as could be judged, the quantity was nicely proportioned to the intensity of the abnormal colour.

But, on the other hand, it may yet be shown that the bronze liver does not belong exclusively to remittent fever. A certain amount of the same kind of colouring matter as that which abounds in remittent fever, is found in many and very variable states of the organ. But, with one exception, I cannot remember to have found it in such quantity as to produce a marked change of colour in the whole liver, unless the patient had had remittent fever. The bronze and deep blue stains on parts in contact with the intestines, rarely penetrate into the hepatic structures beyond the small fraction of an inch. But since this note was commenced, I have become indebted to Dr. McCready for the possession of a liver of the same colour as that seen in remittent fever, and throughout as deeply stained, taken from a person who was not known to have had that disease at any period of his life, and certainly not recently. I saw this man frequently during his last illness, and know his history for the last three or four months. He had for this period a jaundiced hue of the skin, which became intense during the last weeks of his life. He complained of pain in the hepatic region, and of jaundice-pains in his joints and extremities. The liver made a large tumour, filling nearly one quarter of the abdominal cavity, but was smooth on its surface, and its inferior border was regular in shape. This man was a German, and my conversation and inquiries were carried on through an interpreter. He was not asked if he had ever had marsh fever, but he was directed to describe his former sicknesses, and in doing this he gave no account of anything like remittent fever. After death it was found that the common duct was completely obstructed by an adventitious growth within the tube, apparently from its lining membrane; and the tubes above this obstruction were greatly dilated. The liver weighed twelve pounds, and was throughout of a deep bronze colour, except that the enlarged ducts formed, on section, irregular whitish striæ in the otherwise uniform morbid hue. The microscope demonstrated abundance of all the colouring elements found in the liver of remittent fever. The lesion appeared to be essentially the same. It was noticeable in this case, however, that the fine globular hematoïdin had collected unusually in the cells, the globules forming beautiful red groups in almost all the cells, giving them the appearance of certain infusoria fed on cochineal. There was, however, but a single cluster in each cell, and that was commonly near its centre. This could not be bile. It was evidently the same substance that had formed red crystals wholly outside the tubes.

In some cases of intermittent fever, especially that which comes to us from the Isthmus, we see the bronze liver. But this would hardly be claimed as an exception, on account of the known relations between these two affections, and the difficulty of getting an accurate history of diseases that have had a long course before they fall under our observation.

It may be interesting to state that in the last few years I have, in two instances, inspected the livers of persons who had had remittent fever a year and more before their fatal sickness. In both of these the remittent colour still remained, less intense than in recent cases, but yet well marked; and the microscope disclosed the colouring matter unchanged, except perhaps in quantity. Yet these men had enjoyed fair health for twelve months and more, after recovering from the remittent, and one of them had not in that time been exposed to malarious influences. Both died of acute disease, not miasmatic. It would ap-

pear then that this hematoïdin is not susceptible of very ready solution and absorption in the animal fluids. It certainly is not dissolved by water or alcohol, and only slowly and imperfectly by the strong mineral acids. It would further appear, that the deposit is not the disease, but that it is the effect of morbid action in the progress of the disease, and may remain after the fever has subsided without materially affecting the functions of the liver, or greatly impairing the general health.

END OF VOLUME I.

